

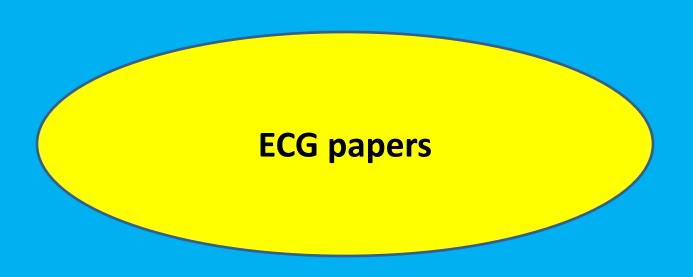
First u have to know basic of ECG Such as

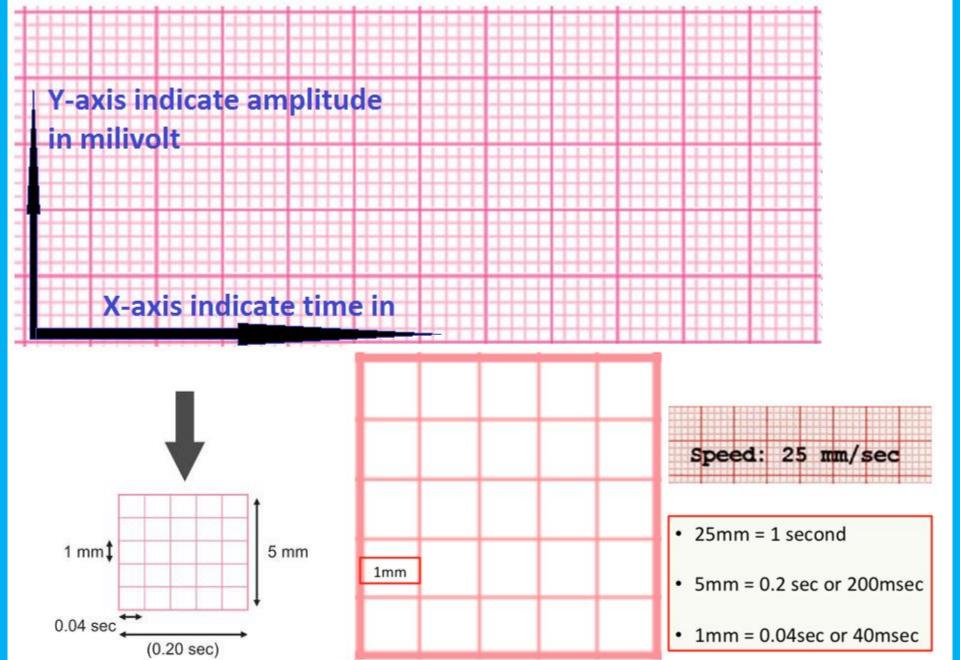
ECG paper

Anatomy of PQRST

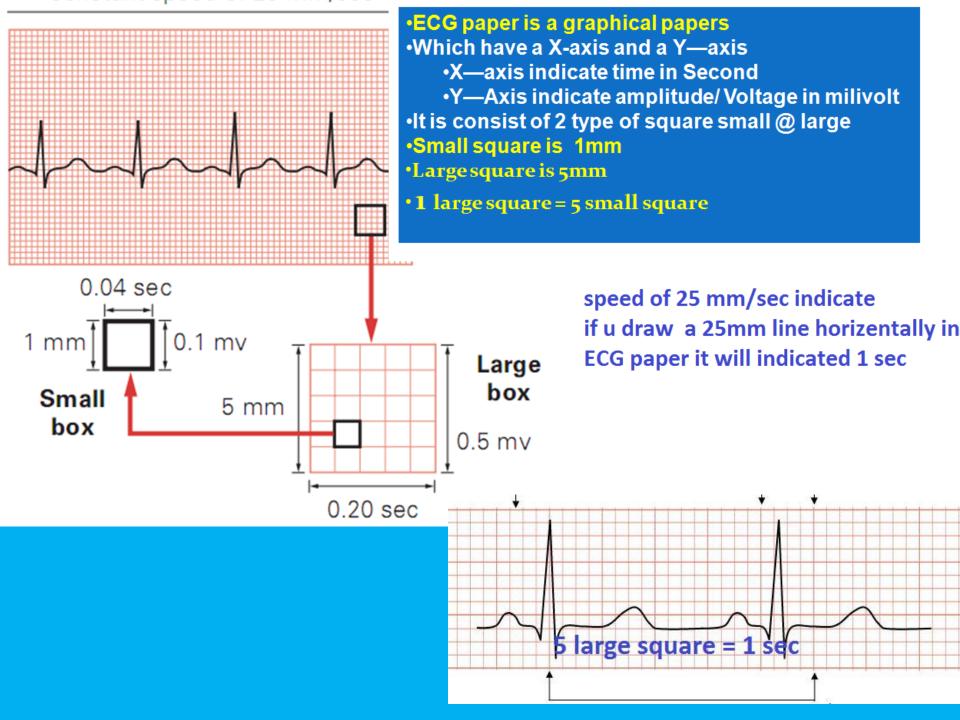
ECG lead

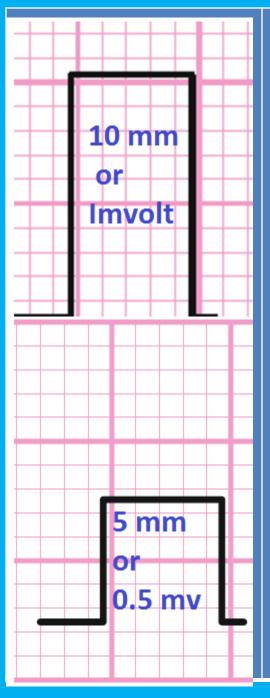
Conduction of electrical pathway





5mm



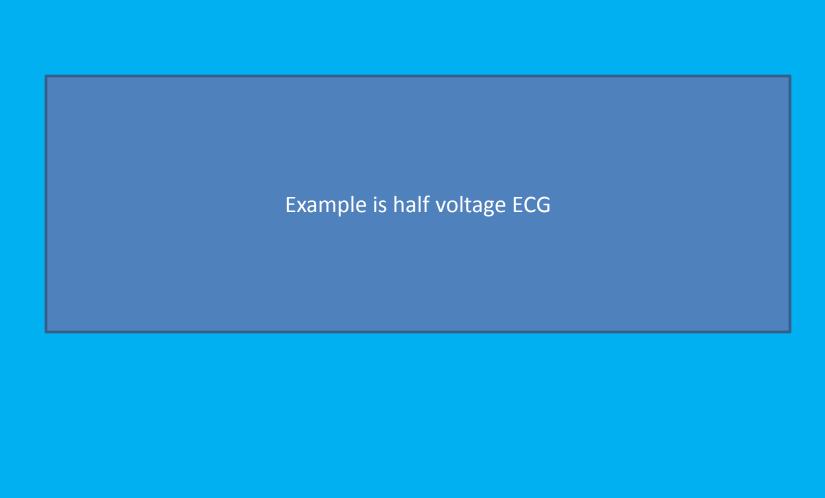


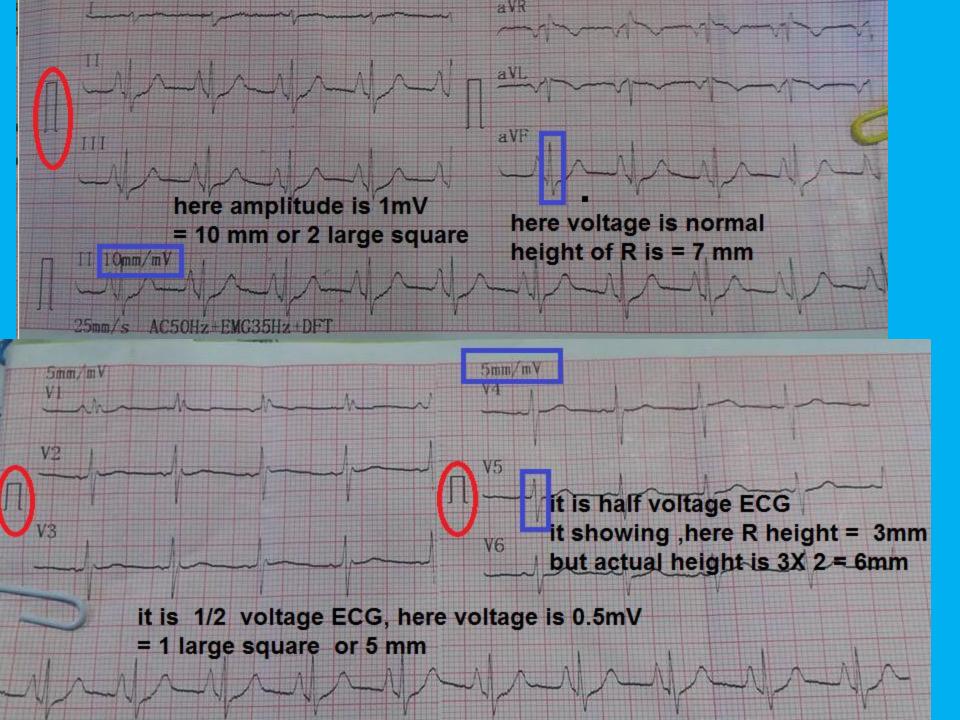
Voltage

Standard voltage of ECG recoding is 1mv 1mv = 10 mm / 10 small square / 2larg square

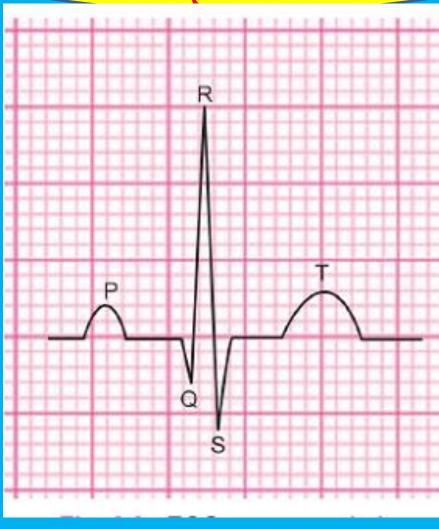
Before interpreting the ECG first look for thevoltage is 2 large square or not

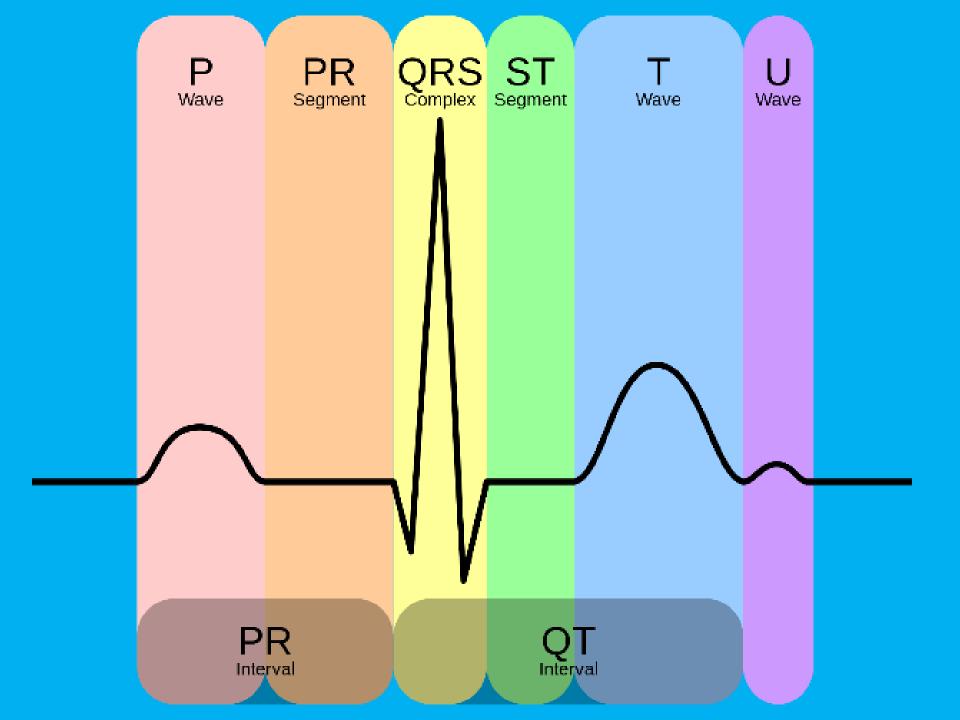
- •If voltage is 1 large square/ 5 small square then the ECG is half voltage ECG
- •Then voltage of P_QRST will Actually be double of what are u seeing



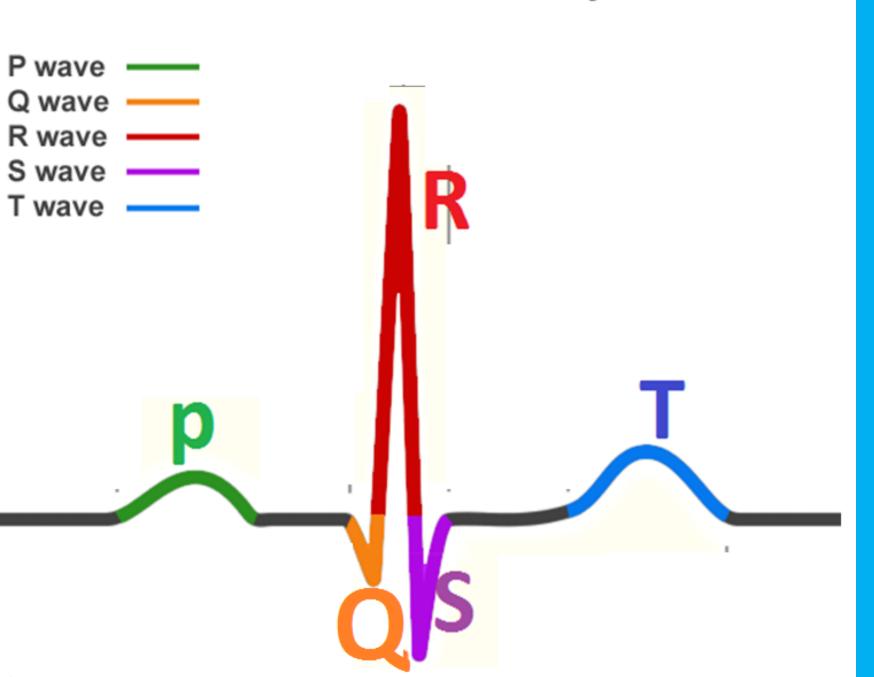


ANATOMY OF PORST

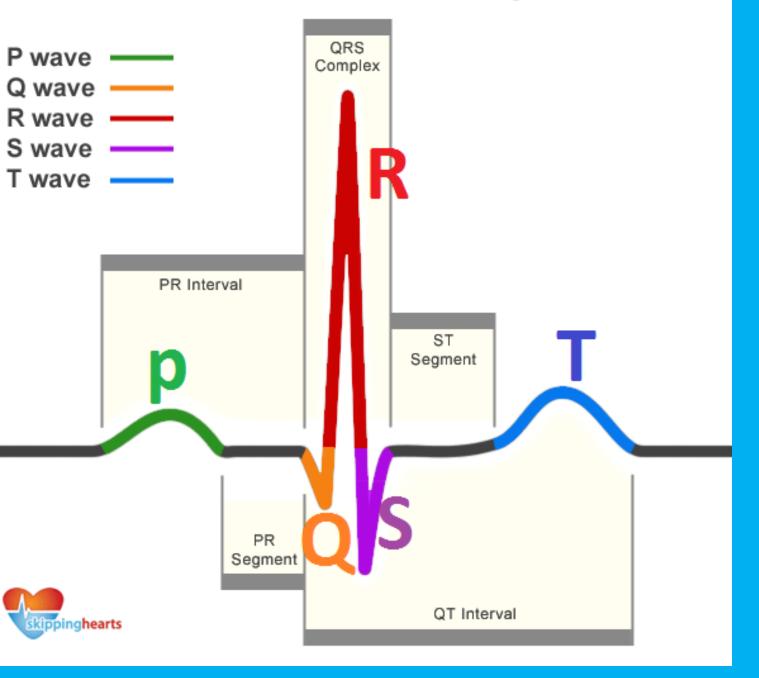


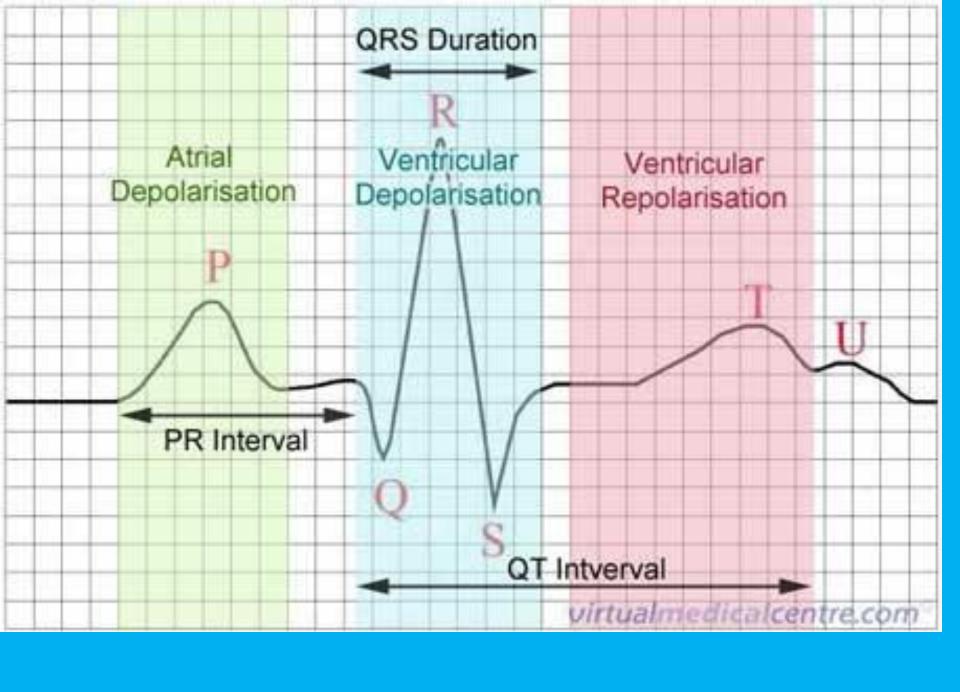


ECG of Normal Sinus Rhythm

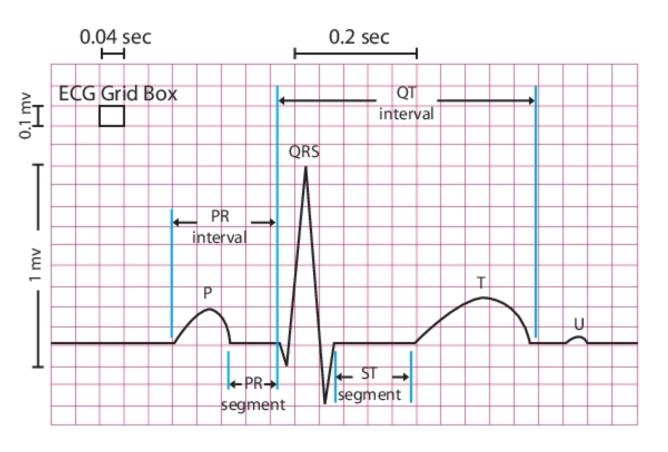


ECG of Normal Sinus Rhythm



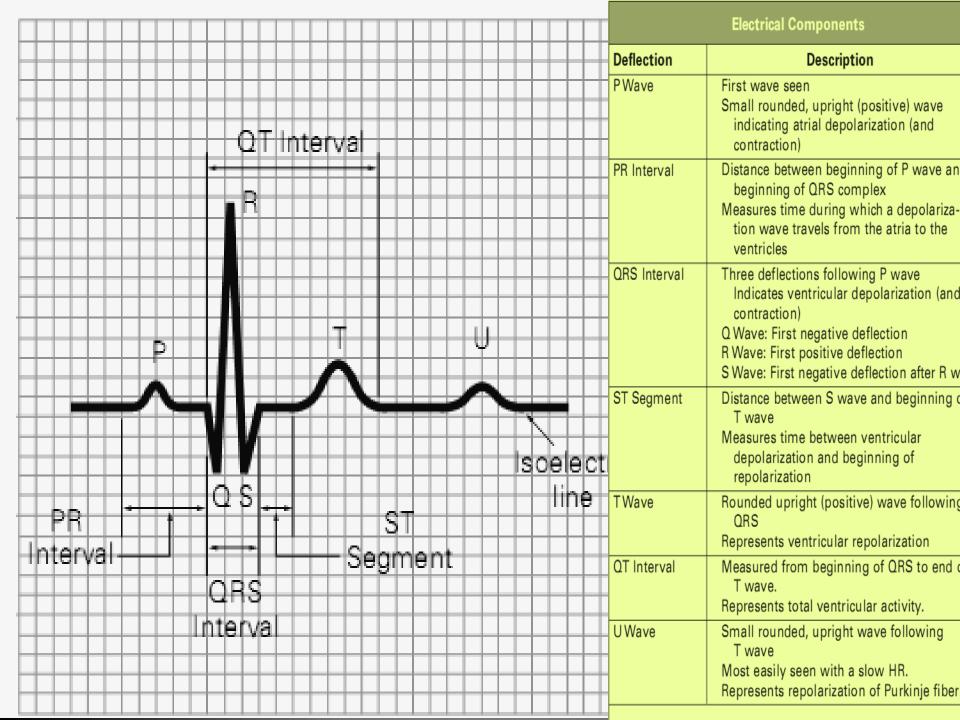


ECG Recording of a Healthy Heartbeat



small square = 0.04 sec
Time large square = 0.2 sec

- PR interval 0.12 0.20 sec •
- QRS duration 0.08 0.10 sec
- QT interval 0.4 0.43 sec
- RR interval 0.6 1.0 sec



Calculation of heart rate

Heart rate

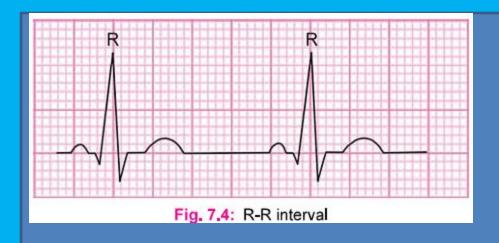
- 1 Small square = 0.04sec
- 1 large square =0.2 sec
- 5 large square = 1 sec
- 1 sec = 5 large square
- 6sec = 30 large square
- 60 second = 300 large square

So heart rate is number of R in 60 sec or 300 large Square

If rhythm is regular

Heart rate = 300 / number of large square in between 2 R

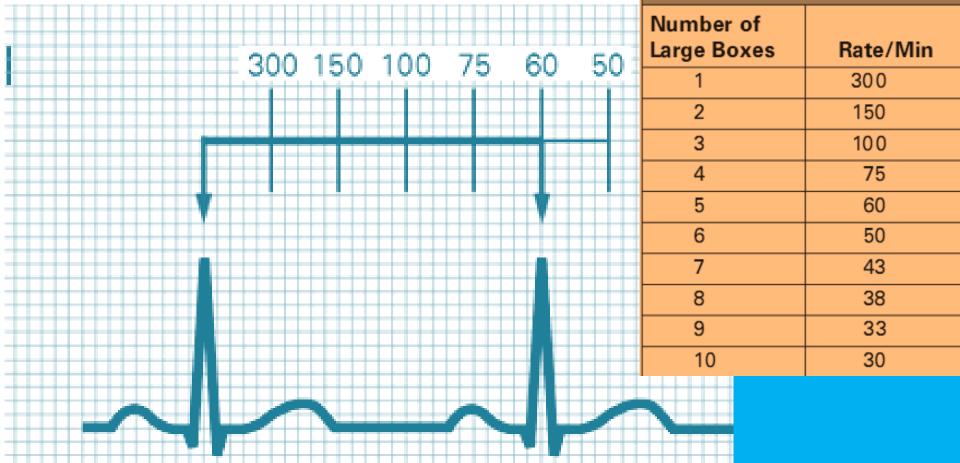
Heart rate = 1500 / number of small square in between 2 R



Here small square between two R is 25 So heart rate is = 1500/25 =60



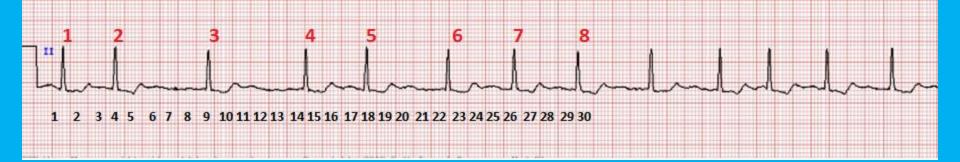
Here large square between two R is 3 So heart rate is = 300/3 =100



If rhythm is regular

It calculate the number of R in 6 sec (30 lagre square) and multiply it with 10

If rhythm is irregular : numbers of R in 30 large square \times 10



```
there is 8 R in 30 large square
So heart rate is = number of R in 30 large square X 10
= 8 X 10
= 80
```



Fig. 7.5: Irregular R-R interval

LEADS

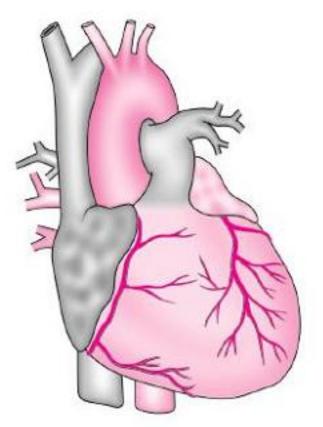
- •Lead is nothing but CC Camera.
- If u place 4 or 6 CC camera in 4 or 6 corner of ur room and u sit in the middle of the room, the camera placed front of u it will take the picture of ur front view and
- the camera placed behind of u will take the post. View of ur body.
- •The side cameras will take left or right lateral surface of ur body.
- Like your self different lead take picture of different part (surface)of the heart such as inferior lead show the inferior surface of the heart

















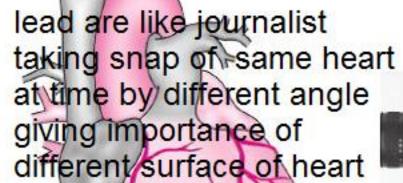








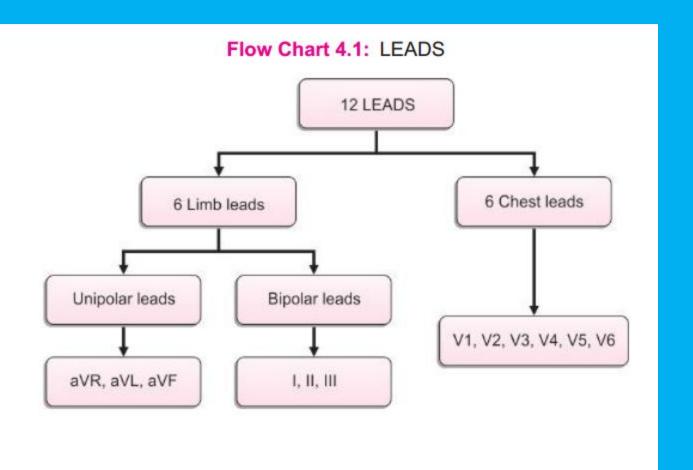


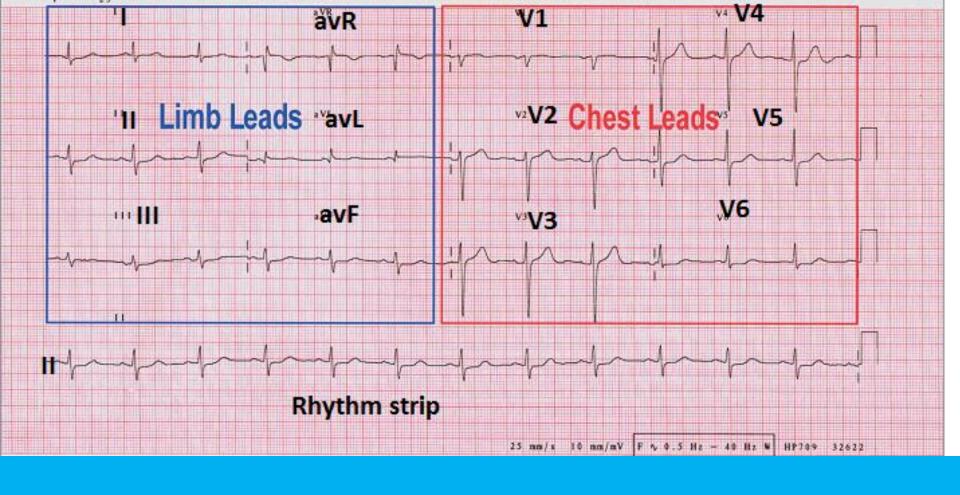


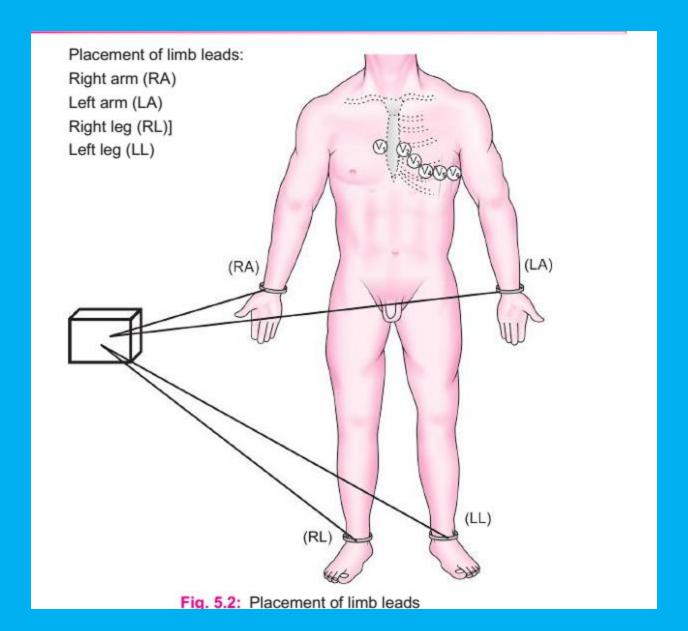












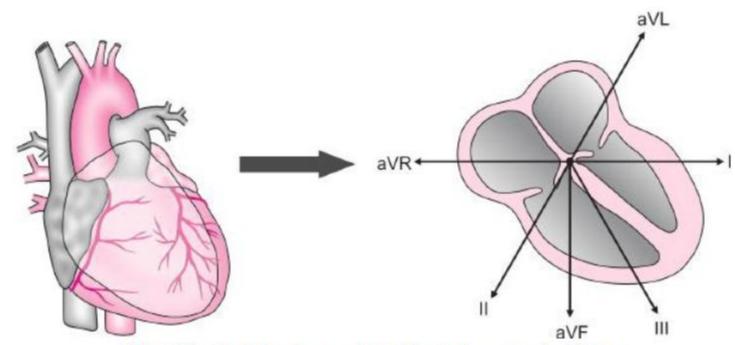


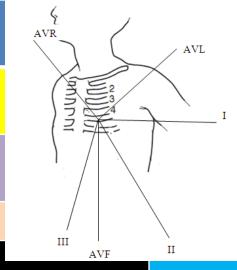
Fig. 4.2: Limb leads are placed in such a way that they bisect the heart at the center in the coronal plane

Here we r seeing that lead I is most close proximity to lead AVL

Lead III is close proximity to lead AVF

Lead II is opposite to lead AVR

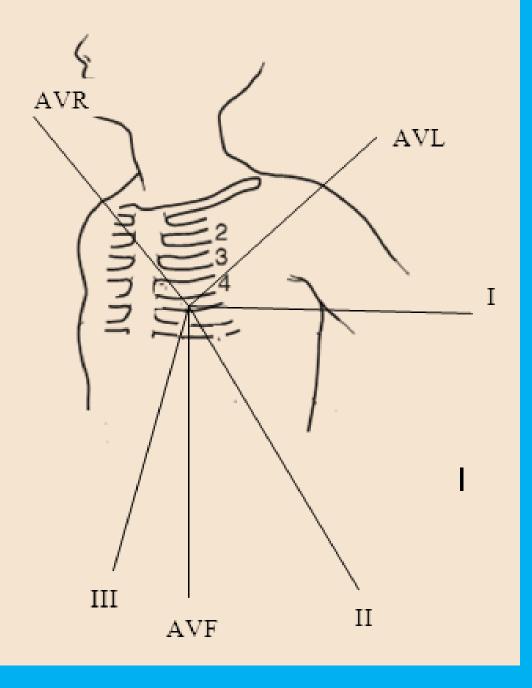
So any change(like T –inversion) in lead I will be found in lead AVL,



what u got in lead II (positive R) will be opposite in lead AVR(negative R)

In this picture we r also seeing that II, III, AVF see the heart from inferior or ground so take picture of the inferior surface of the heart. That s why the are called **inferior lead**

Lead I and AVL are taking the picture from the side of body see the lateral part of the heart that's why lead I and lead AVL are called **Lateral lead**



So

I = AVL

III = AVF &

II # AVR

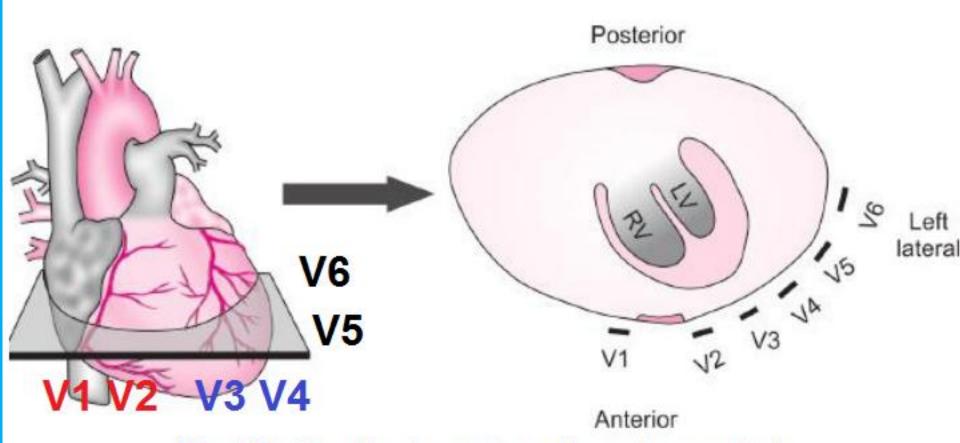
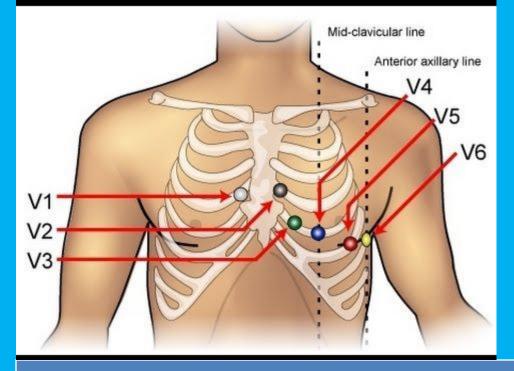


Fig. 4.3: Chest leads are placed in such a way that they bisect the heart in the horizontal plane

These lead see the heart at horizontal plan Leads V1 and V2 look at the right ventricle; leads V3and V4 look at the septum; leads V5 and V6 look at the left ventricle



V1: RT 4th intercostal space

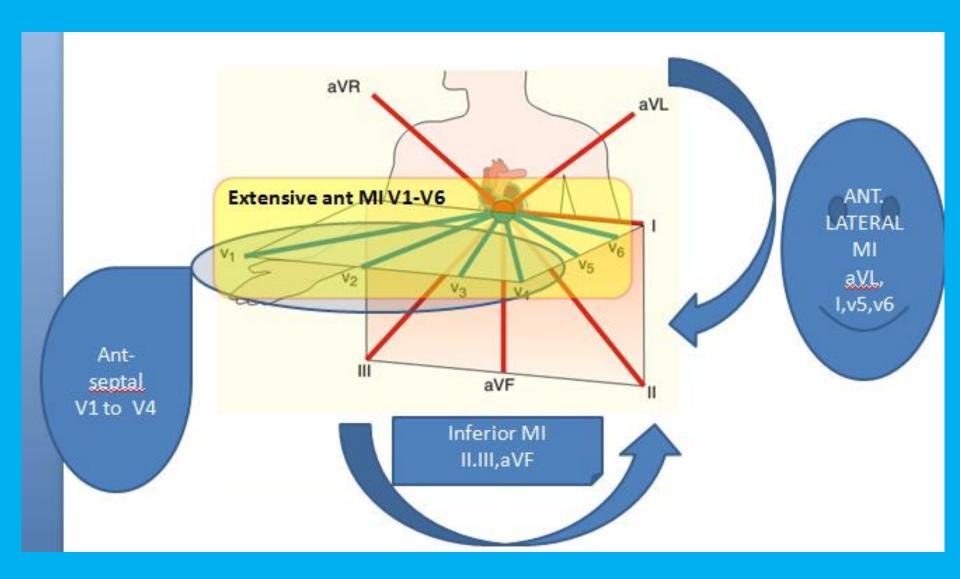
V2: LT 4th intercostal space

V4: LT 5th intercostal space in midclavicular line

V3: halfway between V2 and V4

V5: LT anterior axillary line at the same level of V4

V6: LT midaxillary line at the same level of V4 & V5



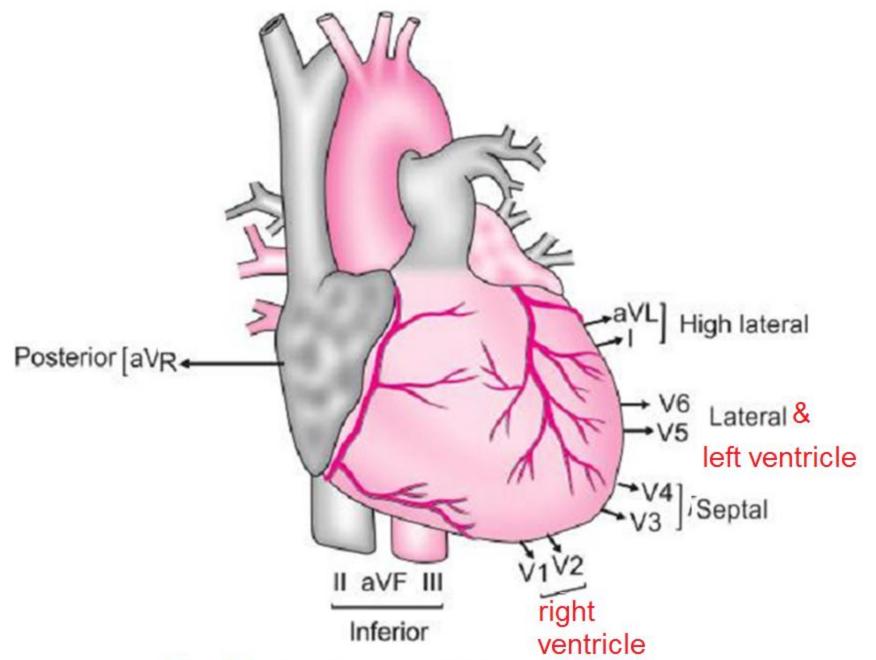
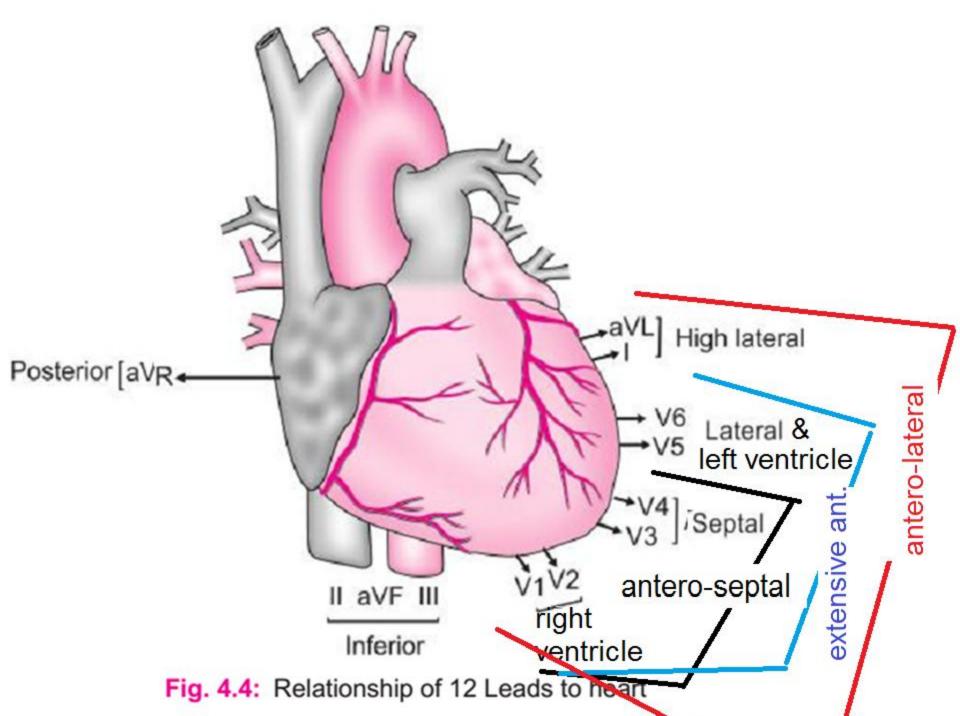
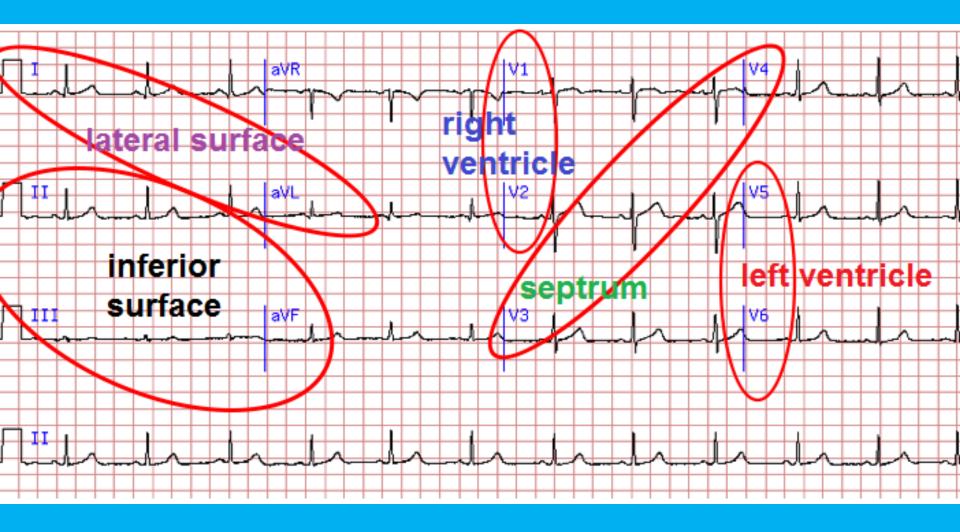
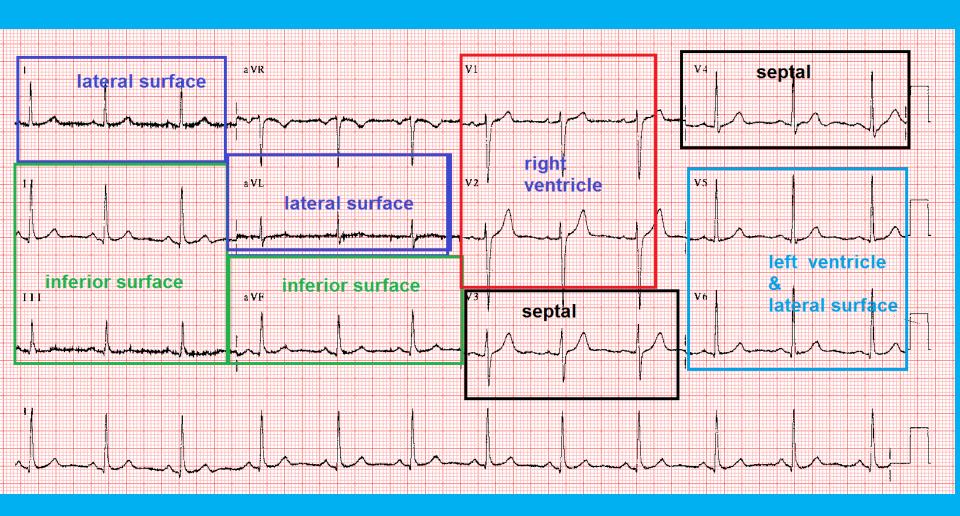


Fig. 4.4: Relationship of 12 Leads to heart







EMSDoc911	Leads	Artery	Presentation	Reciprocal Changes
Ant. Septal	Clocold Hollymanood MI	None		
extensive	V1-V6	LAD	crushing CP, diaphoresis	
anterior				Inferior
Inferior	II, III, aVF	RCA	1) Epigastric Pain, N/V 2) Syncope 2/2 bradyarrhythmias from SA node involvement	Lateral
Lateral	I, aVL	LCX		Inferior
Posterior	V1, V7-V9	PDA 80/20% from RCA/LCX	Subtle signs, non-descript CP	Ant. Septal
R Ventricle	V1, V4R	RCA	Hypotension, can be associated with Inferior AMI	Lateral
*LAD – L ant descer *PDA – post. descer	EMSDoc911			

RATES OF PACEMAKERS

1. SA node

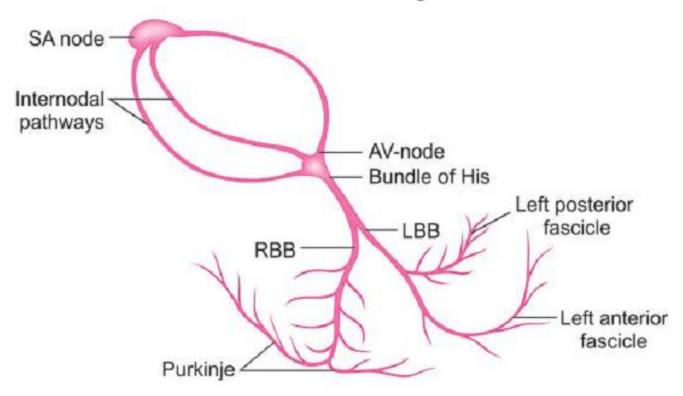
2. Atrial cells

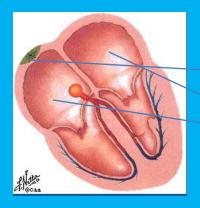
3. AV node

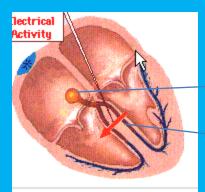
60 - 100 bpm

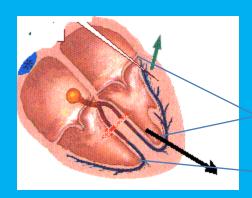
55 - 60 bpm

45 - 50 bpm









SA node ----

■Right atrium----



■AV node ----

■bundle of HIS ---

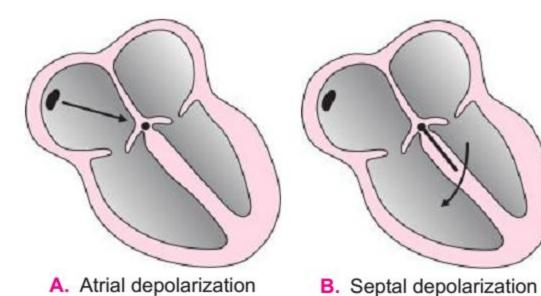


Then left ventricle (as mass of left is more than right net vector is toward

Right venticle

the left)---

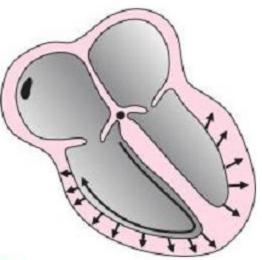
At last post and basal part of the heart



NORMAL SPREAD OF ELECTRICAL ACTIVITY IN THE HEART

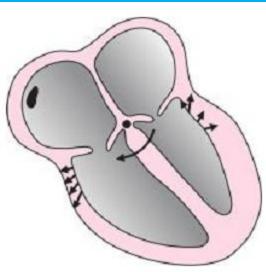


 Depolarization of anteroseptal region of the ventricular nyocardium

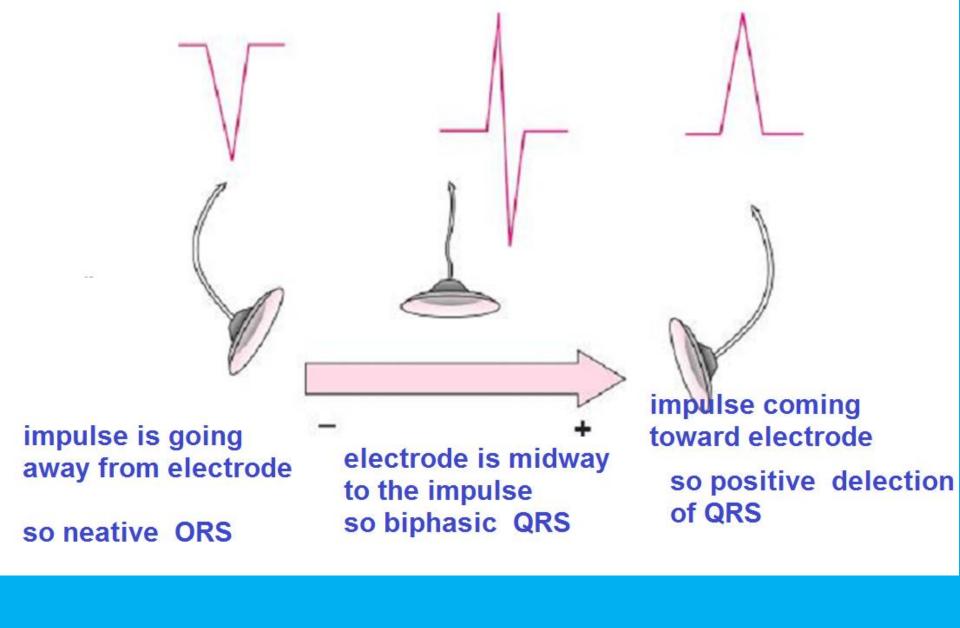


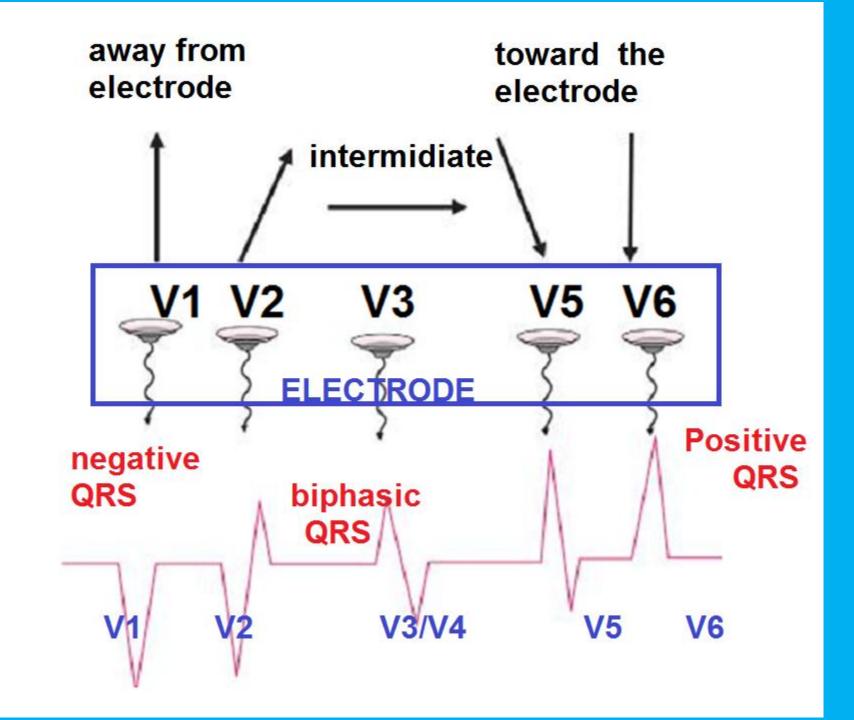
from left to right

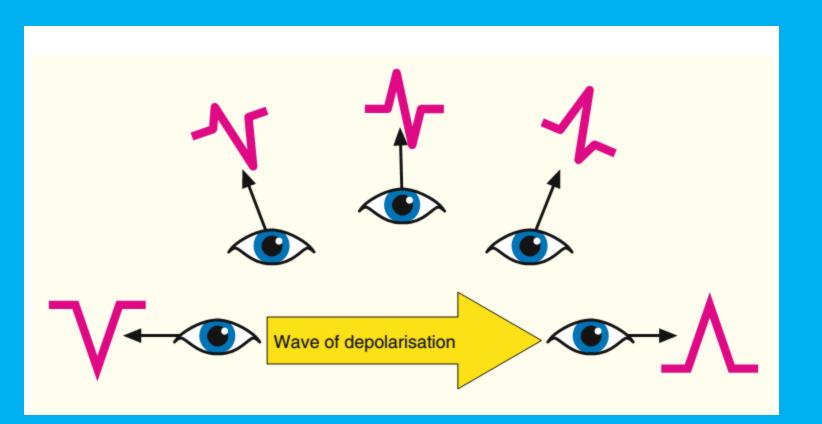
D. Depolarization of major portion of ventricular myocardium from endocardial surface to epicardium



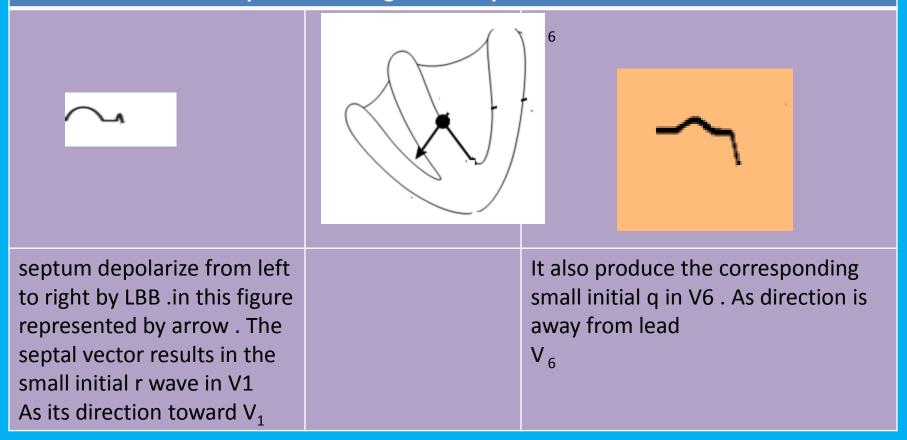
E. Late depolarization of posterobasal portion of the left ventricle and pulmonary conus



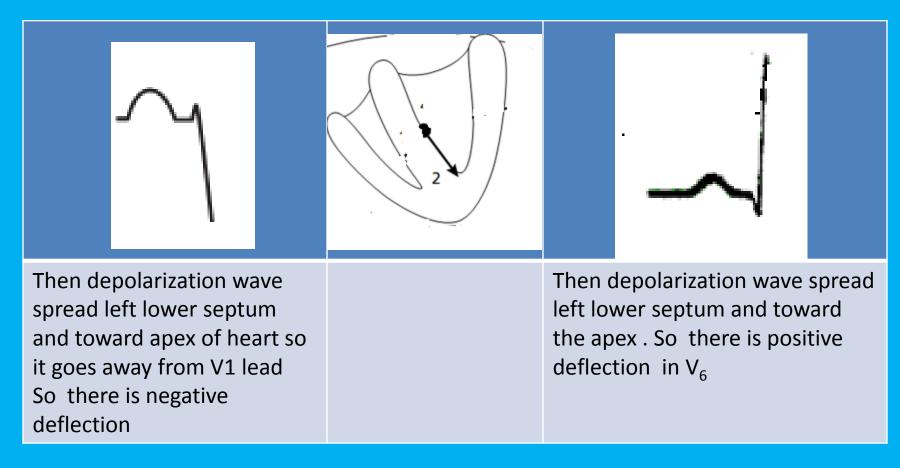


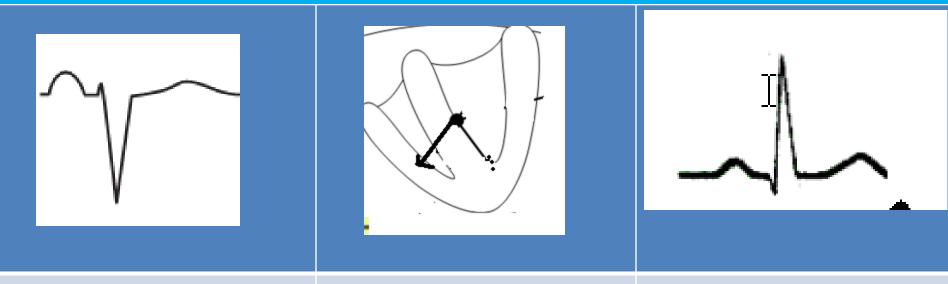


Depolarization of SA node to atrium cause P wave . Depolarization of AV node cause PR Interval. Ventricular deploariztion begin with septum



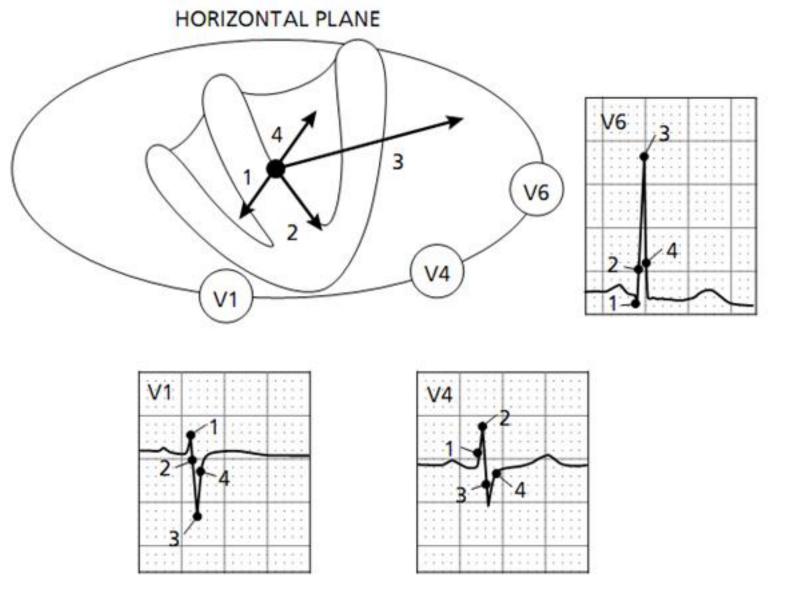
Then depolarization wave spread left lower septum and toward apex of heart so it goes away from V1 lead and toward the V





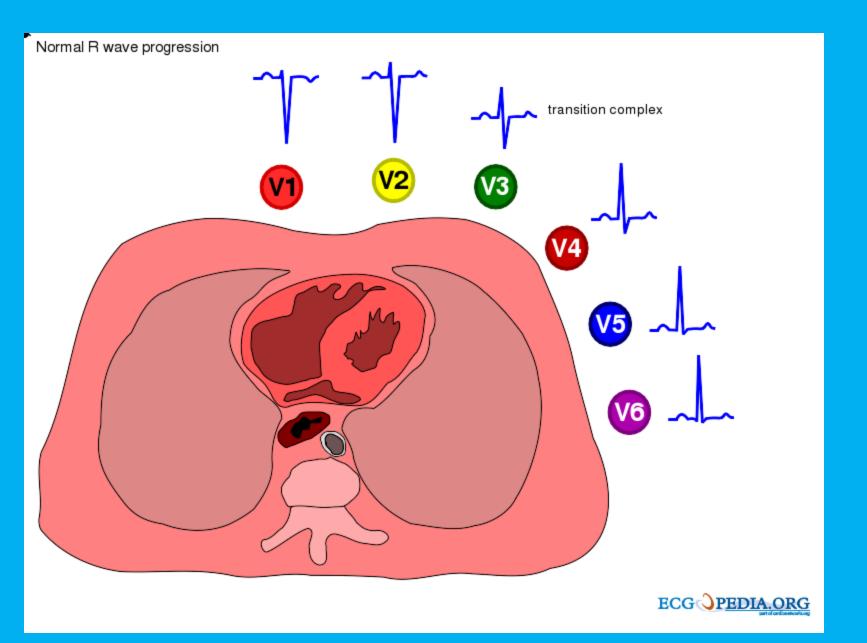
Now impulse goes toward d right ventricle and so in V1 ECG line goes up ward come to base line

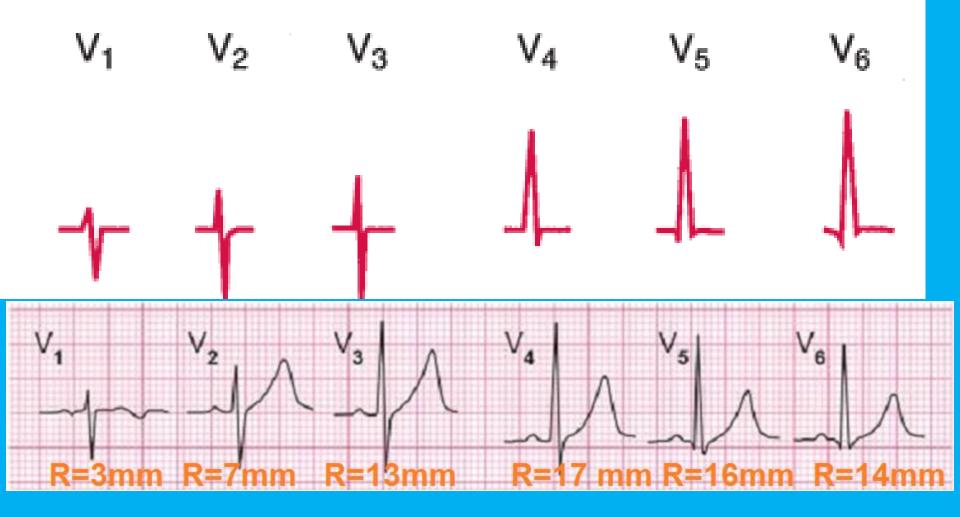
To V-6 it wil be away from the lead . So positive wave goes down ward



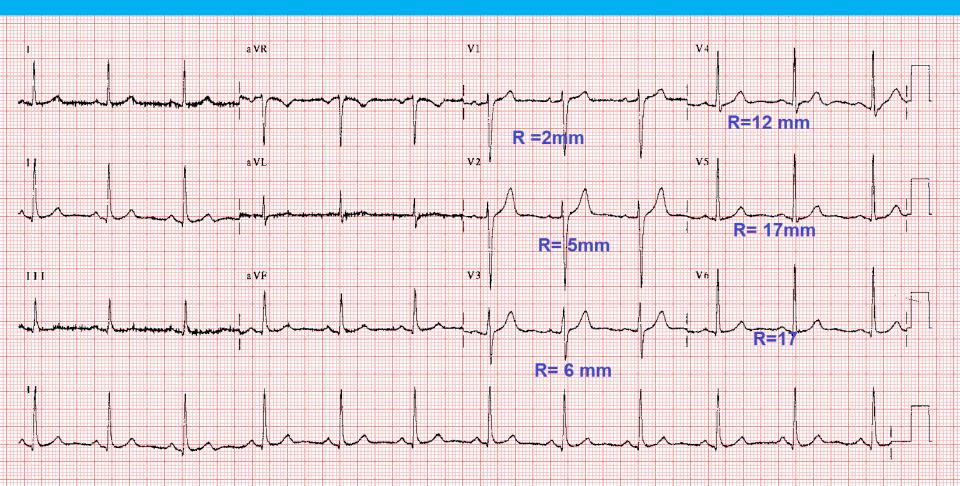
So this cause why there is progressive increasing length of R wave from V₁ to V₆

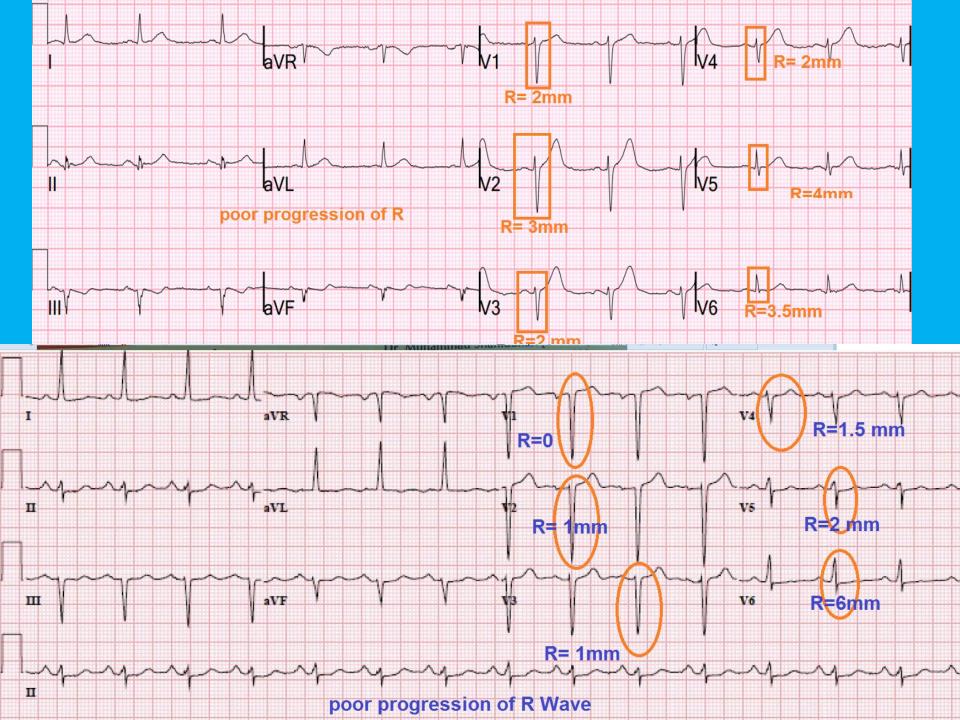
SLOW PROGRESSION OF R WAVE



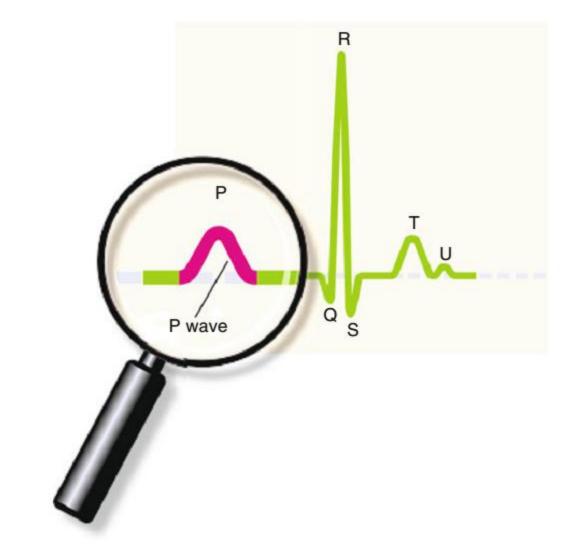


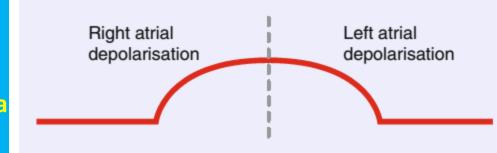
Progressively increasing the height of the R wave from V1 to V6





wave





P wave --- Indicate depolarization of atria

Anatomy of P wave

- Height and wide: 2.5 X 2.5 mmP may be
 - Normal
 - Tall
 - Notch / wide
 - Absence



Cause of absent p

- Atrial fribrillation ave
- Atrial flutter
- SVT
- •VT
- Ventricular ectopic
- Hyper kalaemia

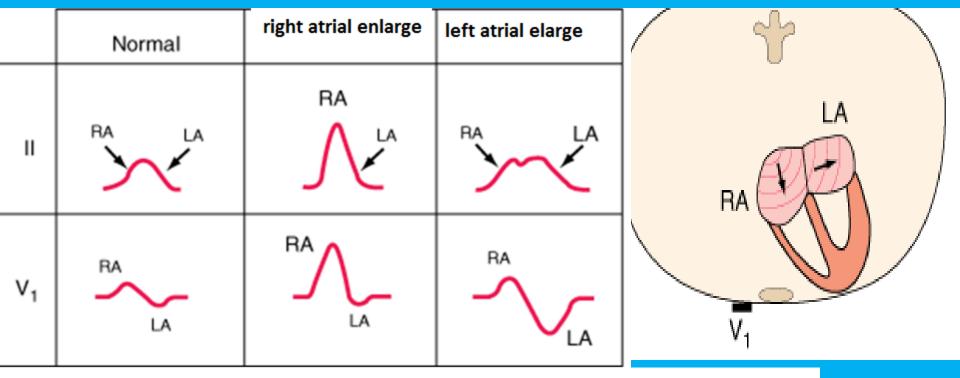
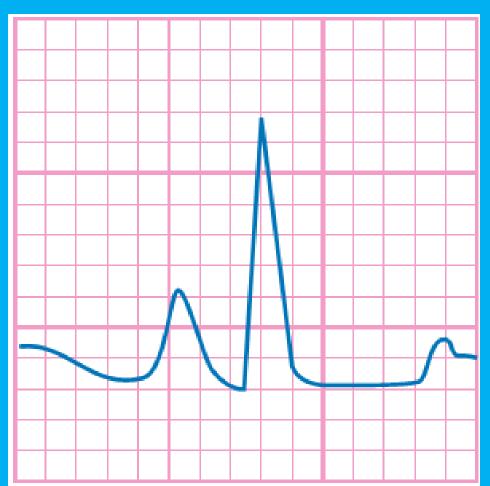
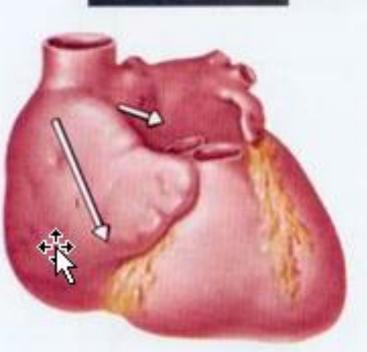


Table 5.1 P wave changes in leads II and V1, occurring with atrial abnormalities

Abnormality	Lead II	Lead V ₁
Right atrial abnormality		
Left atrial abnormality		
Bilateral atrial abnormality		

L --- pulmonale







Right atrial enlargement



Right atrial enlargement:

lead 2 shows P amplitude >3 mm; in V1, P is biphasic, the first half of the P wave is positive and >1 mm wide



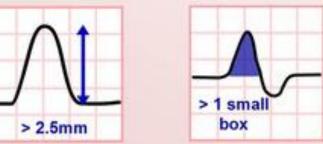




Fig. 7.12: P pulmonale

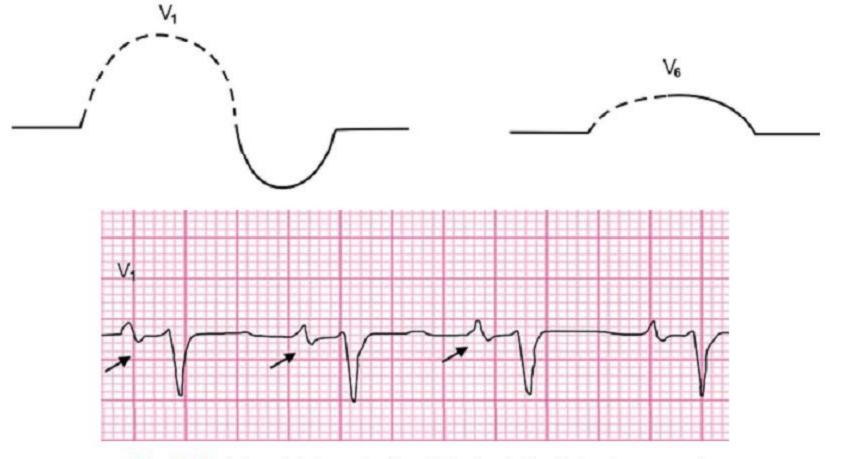
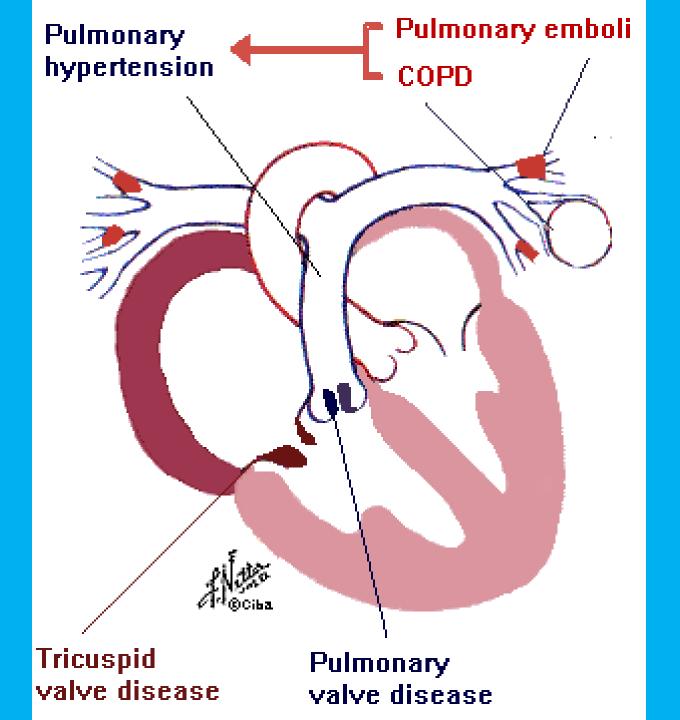
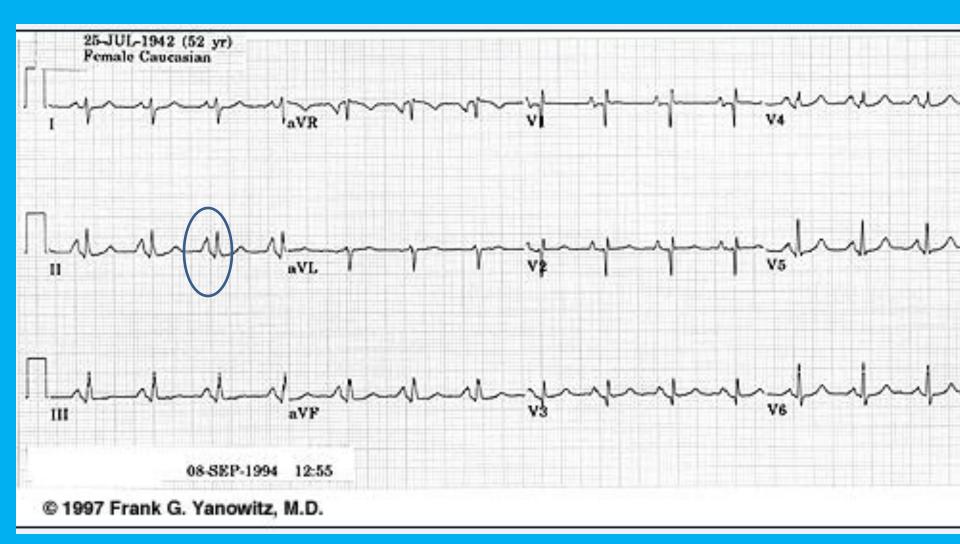
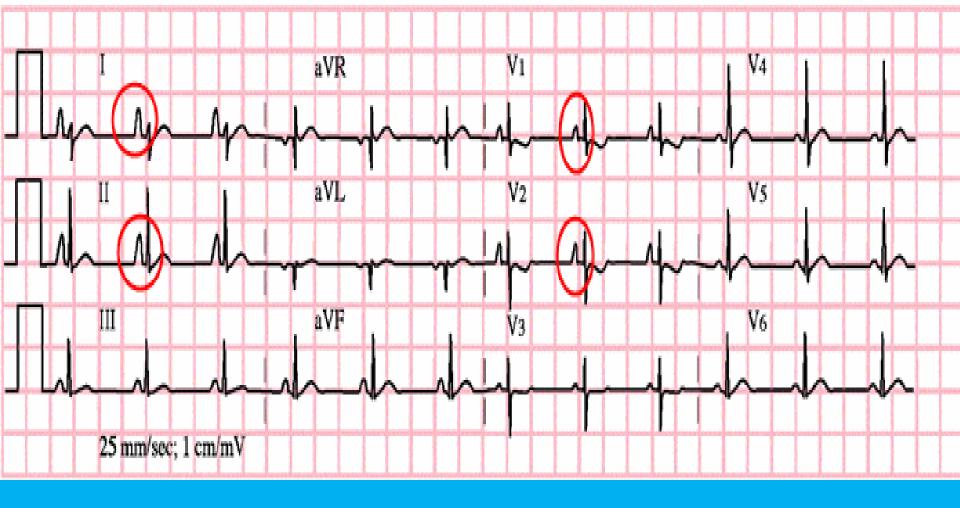
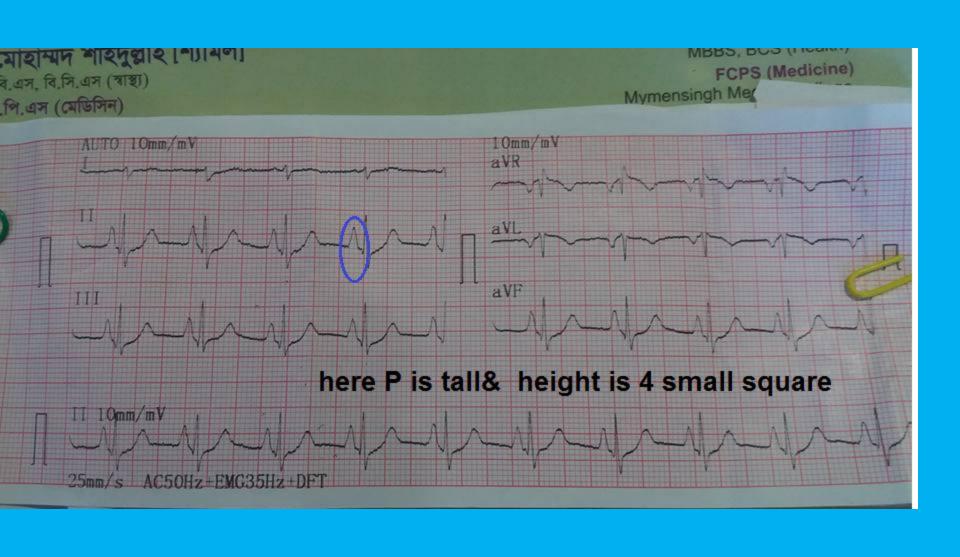


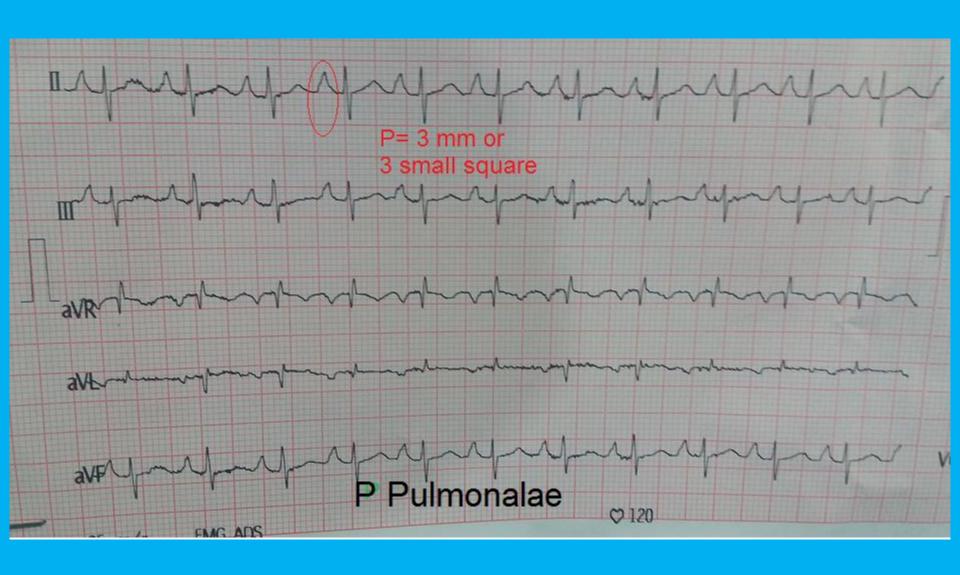
Fig. 7.13: Intra-atrial conduction delay in right atrial enlargement











P----Mitrale

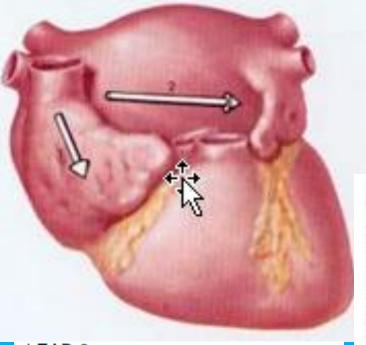






Fig. 7.11: P mitrale

LEAD 2

Left atrial enlargement



LEAD V₁

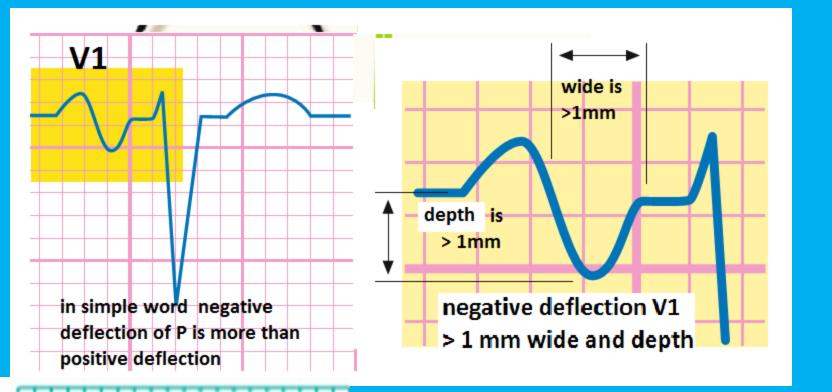
Left atrial enlargement

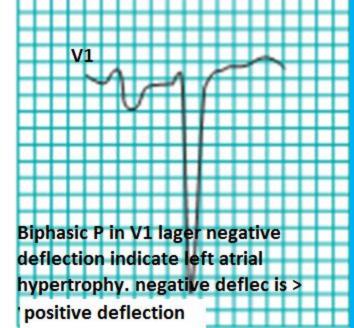


Left atrial enlargement:

lead 2 P wave duration greater than three small squares (0.12 second);

lead V1 the negative component of the P wave occupies at least one small box:





In V1 if the second half of P wave is wider and deeper than 0.4 second (1 mm), then it is likely to be left atrial enlargement.

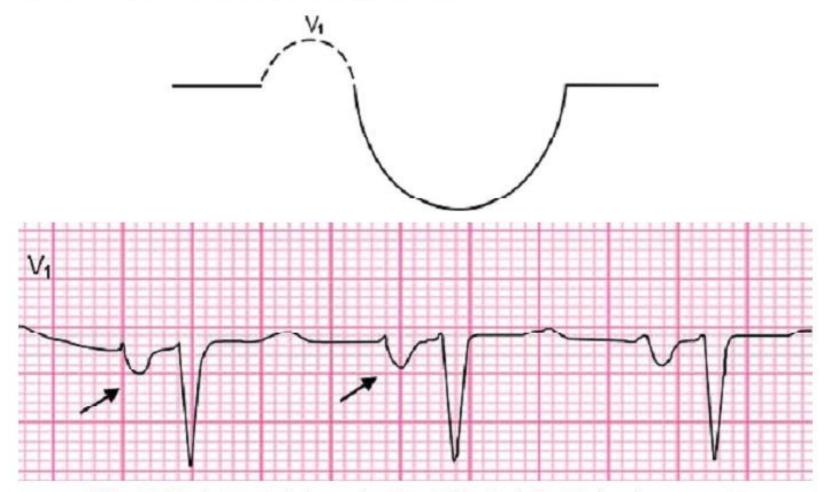
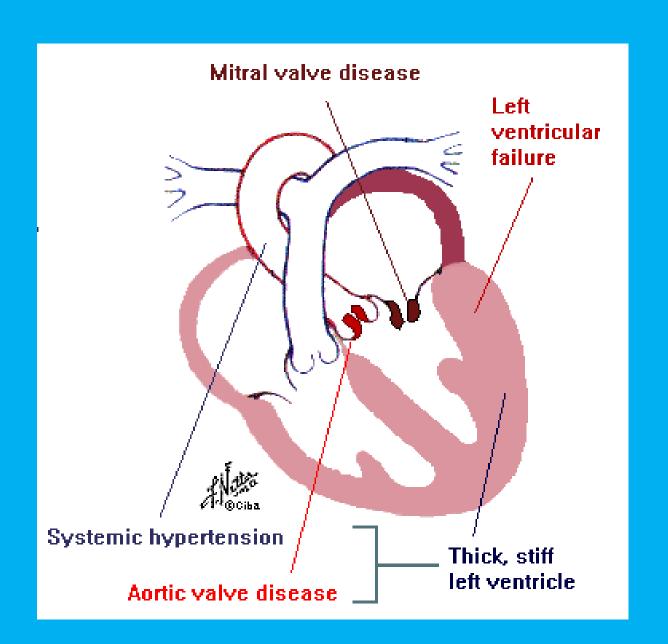


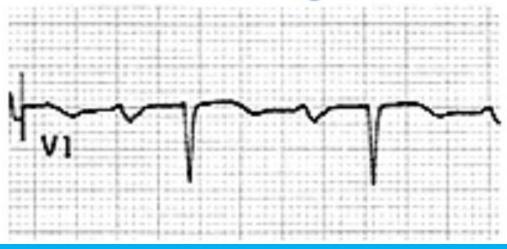
Fig. 7.14: Intra-atrial conduction delay in left atrial enlargement



Lead II



Lead V_I



Question regarding P-Wave

Cause of absent p wave

- Atrial fribrillation
- Atrial flutter
- •SVT
- •VT
- Ventricular ectopic
- Hyper kalaemia

Wide P wave:

- 1. Left atrial hypertrophy or enlargement Tall P wave:
- 1. Right atrial hypertrophy or enlargement Small P wave:
- 1. High nodal rhythm
- 2. High nodal ectopic
- 3. Atrial tachycardia
- Atrial ectopics

Inverted P wave:

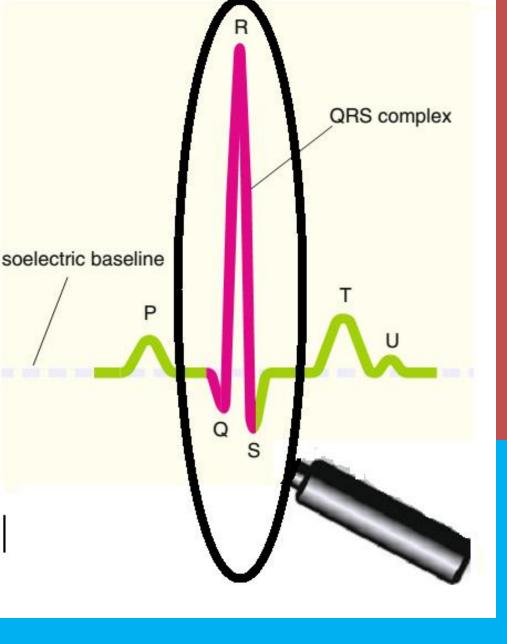
- 1. Nodal rhythm with retrograde conduction
- 2. Low atrial and high nodal ectopic beats
- 3. Dextrocardia

Variable P wave morphology:

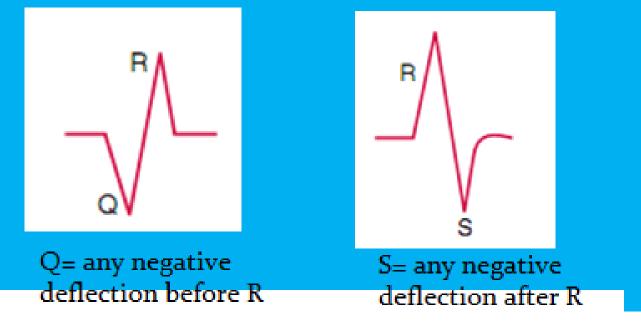
Wandering pacemaker

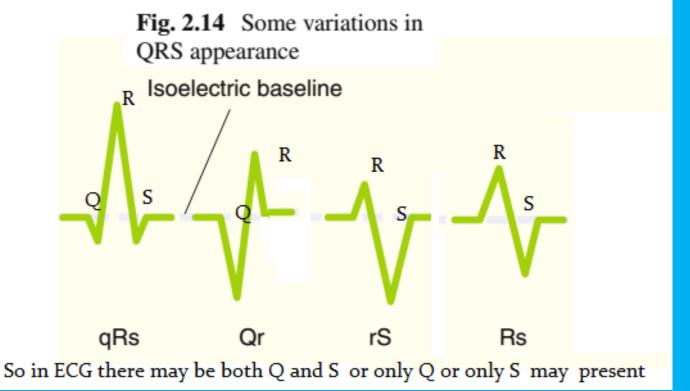
Multiple P waves:

1. Third degree heart block



R wave QRS complex:





R wave may

Tall –ventricular hypertrophy

Small – low voltage ECG

Poor progression ----MI

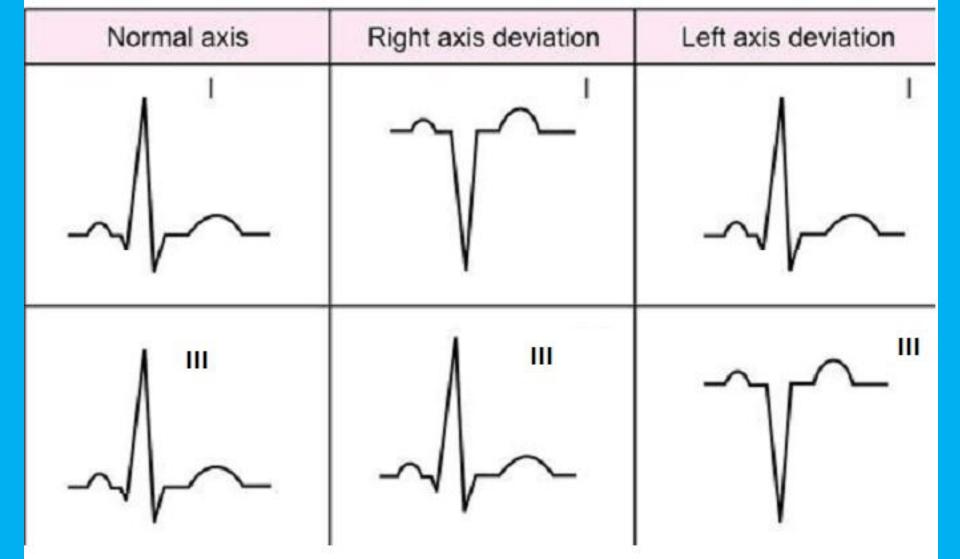
Abnormal R or QRS complex---RBBB, LBBB, VT, SVT, VF

R wave also use in Determination of Axis

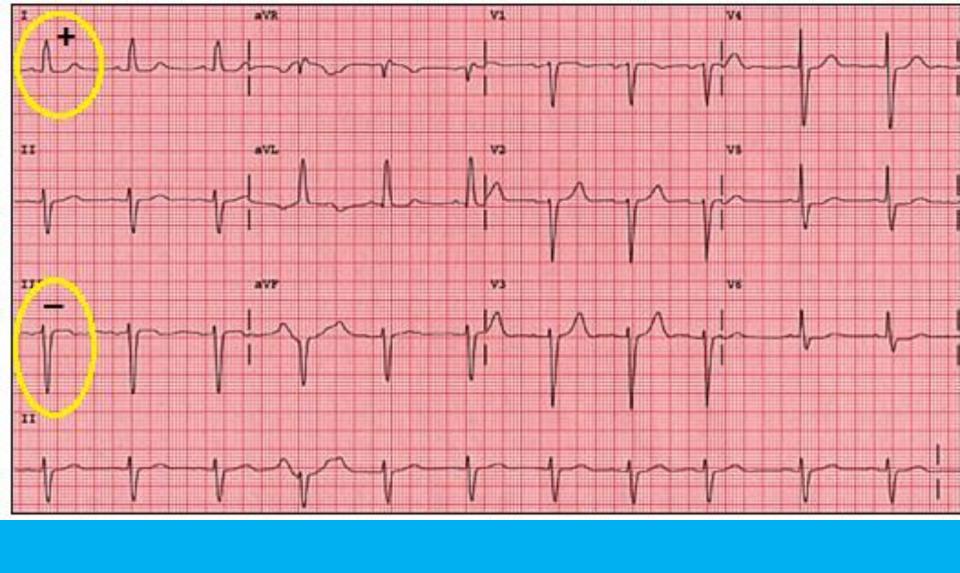
AXIS DEVIATION

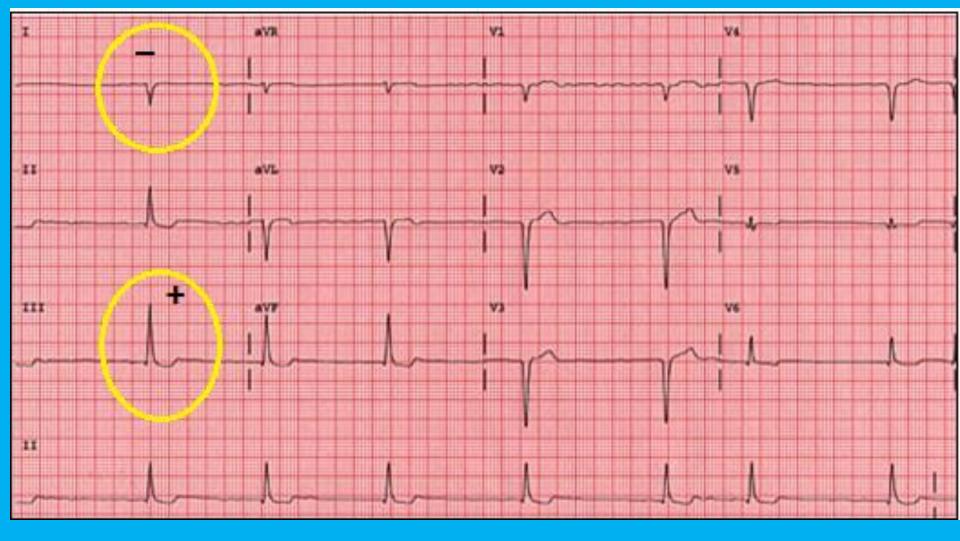
	QRS—in Lead I	QRS —in Lead III / AVF
Normal axis	Positive (+)	Positive (+)
left axis deviation	Positive (+)	Negative (-)
Right axis deviation	Negative (-)	Positive (+)
Indeterminate axis	Negative (-)	Negative (-)
Positive mean	Negative mean	
QRS complex upward;	QRS complex downward	

Right axis deviation	Left axis deviation
Right ventricular hypertrophy	Left ventricular hypertrophy
Right bundle branch block	Left bundle branch block
Left posterior hemiblock	Left anterior hemiblock
Emphysema and cor pulmonale	Wolf-Parkinson-White syndrome
Fallot's tetralogy	Hypertrophic cardiomyopathy
Inferior MI	Lateral MI









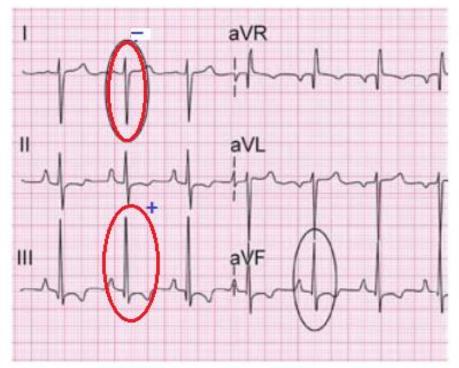
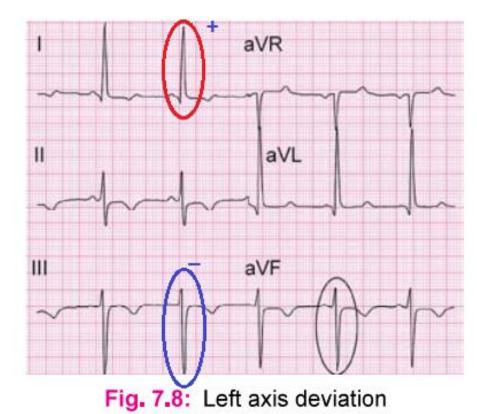
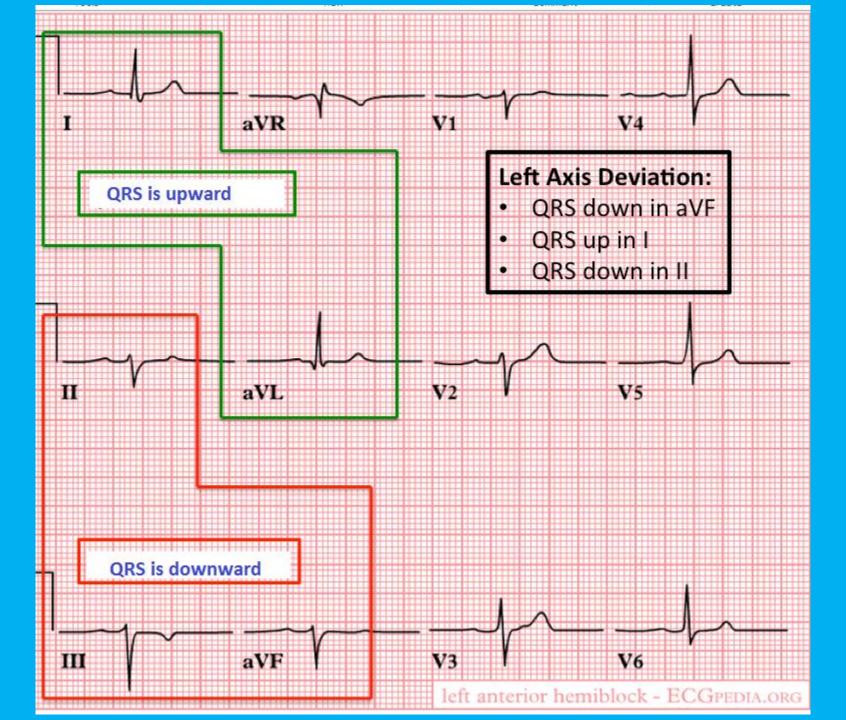
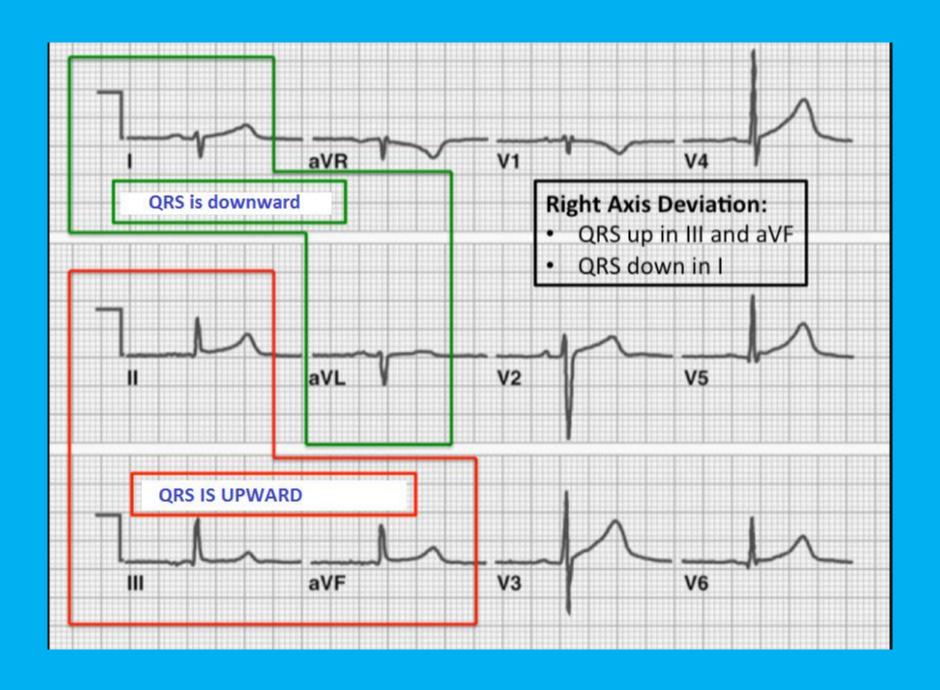
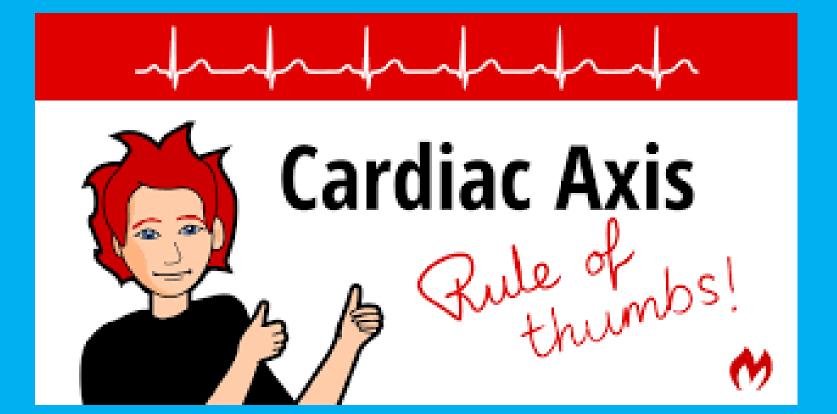


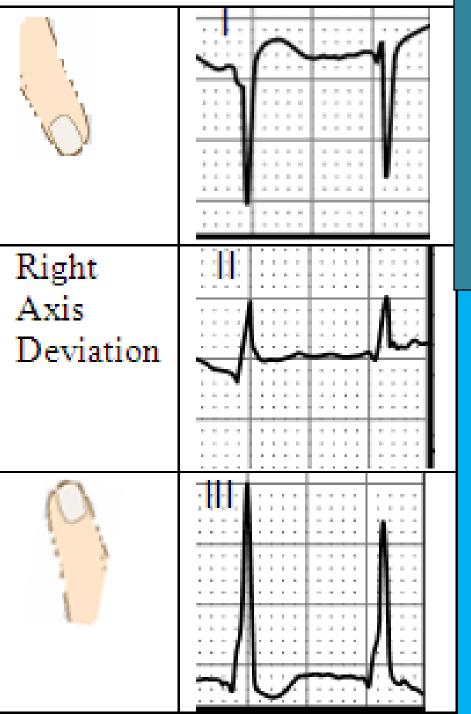
Fig. 7.7: Right axis deviation







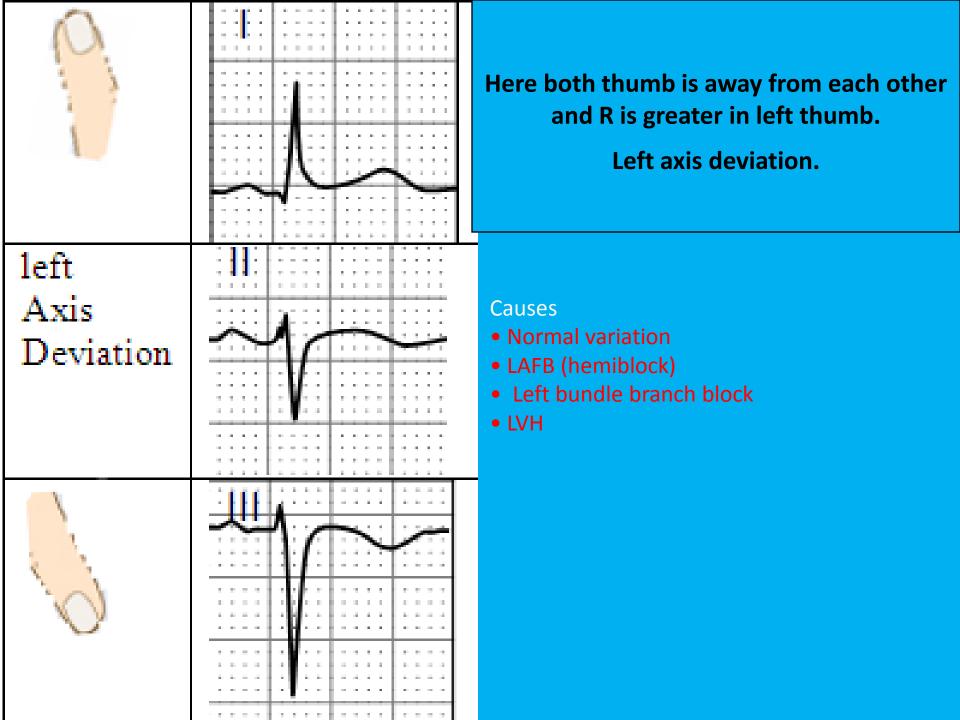


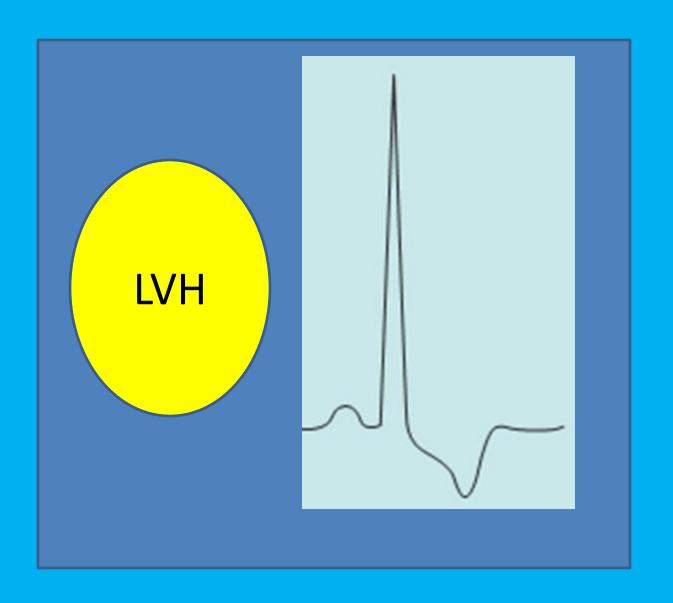


Here both thumbs are directed toward each other. And R is greater in right (iii)

Causes

- Normal variation
- RVH
- LPFB
- Lateral MI
- Pulmonary embolism
- Dextrocardia





Left ventricular hypertrophy (LVH)

Left ventricular hypertrophy (LVH) if full field of the following:

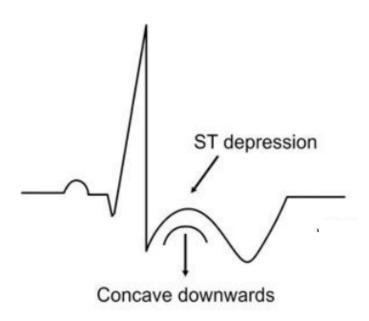
R in
$$V_5/V_6 > 25$$
 mm

R in
$$V_5/V_6 + S$$
 in $V_1 > 35$ mm

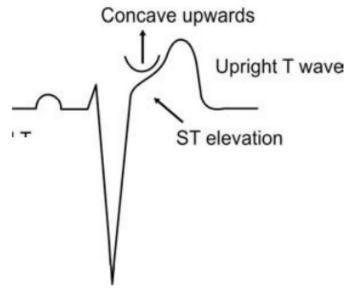
R in AVL > 13

R in AVF > 20

if LVH with T inversion or ST depression (I,AVL, \vee 4 to \vee 6)= LVH with strain



In V4,V5,V6 there in ST depression with concavity downward & asymmetrical T inversion

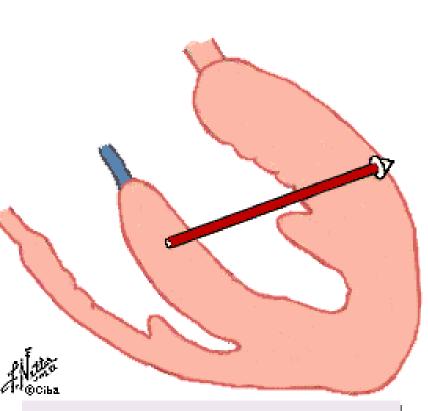


In V1,V2,V3 there is ST elevation with concavity upward and T is up right and asymmetrical these are reciprocal change of V5,V6

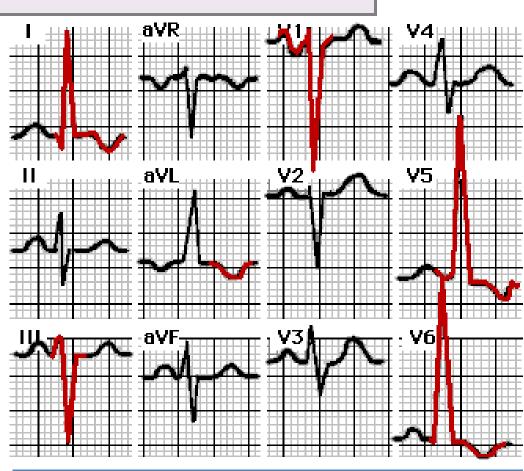
a, a, aect. 2, ca, a or o

Left Ventricular Hypertrophy (LVH)

High voltage in limb leads: (R I + S III >25 mm)
Or precordial leads: (S V1 + R V5, or S V1 + R V6, >=35 mm)
Often, left atrial enlargement, ST-T abnormalities



Arrow indicates major electrical vector of ventricular depolarization

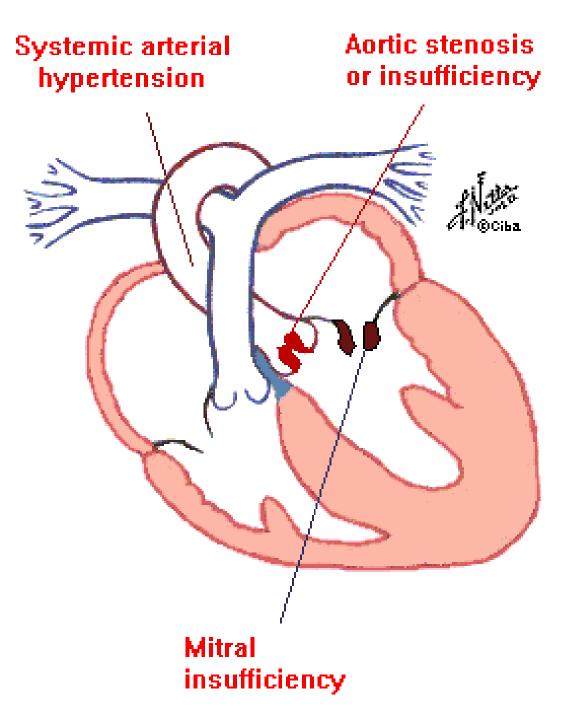


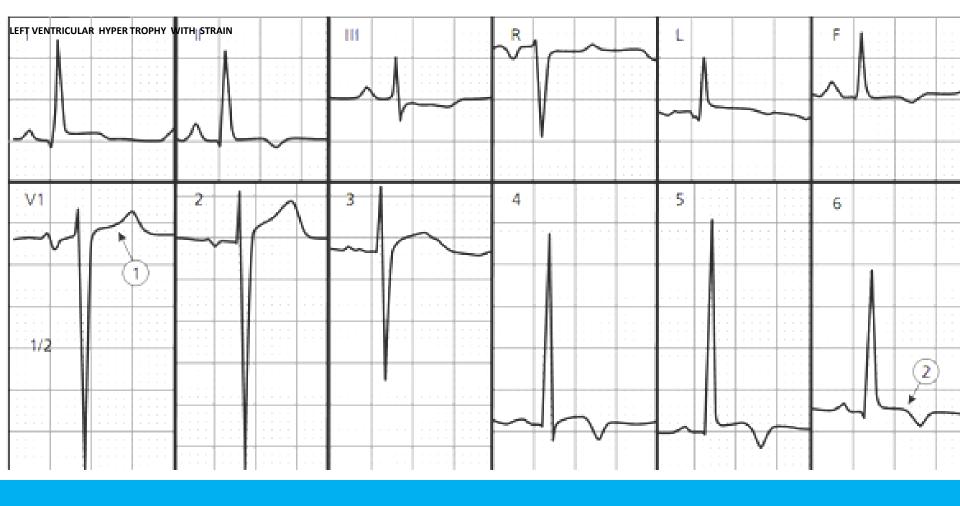
Click to go to Right Ventricular Hypertrophy

Cause of LVH

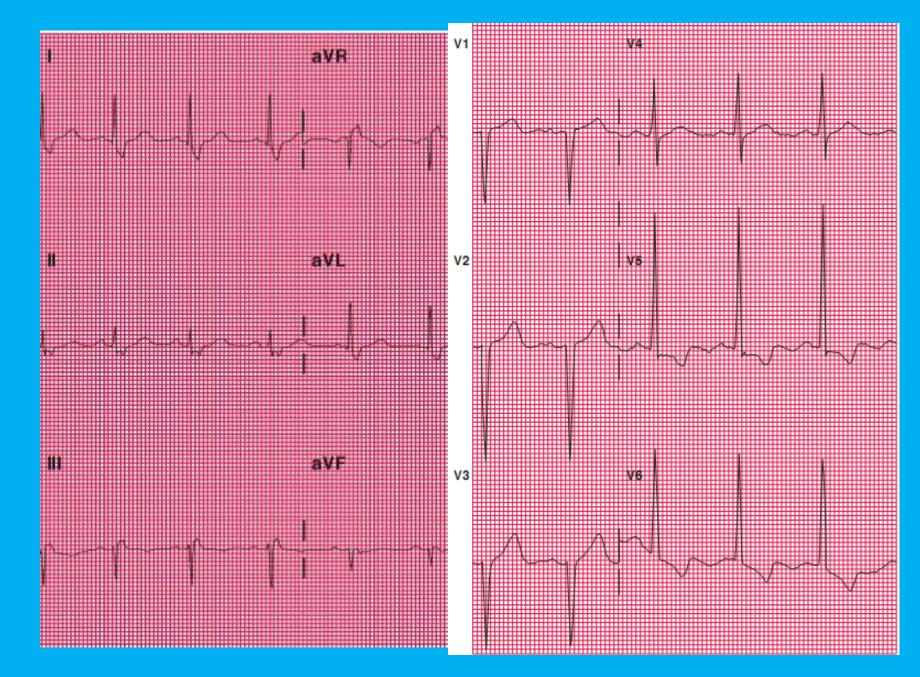
- **1.HTN**
- 2. Aortic stenosis
- 3.**AR**
- 4.**MR**
- 5. Hypertrophy cardiomyopathy
- 6. Corctation of aorta

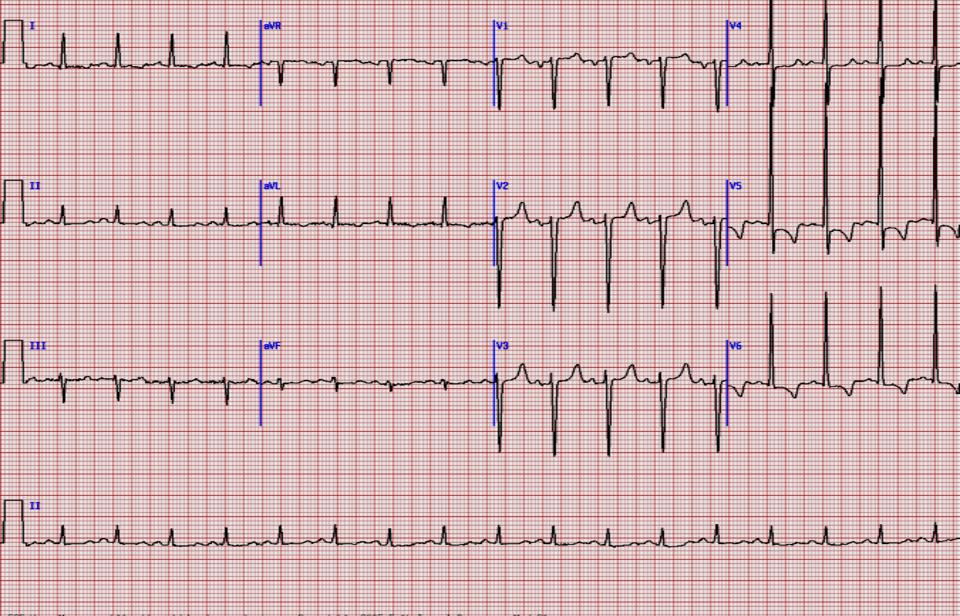
Confirm by Echo and Apex beat not shift

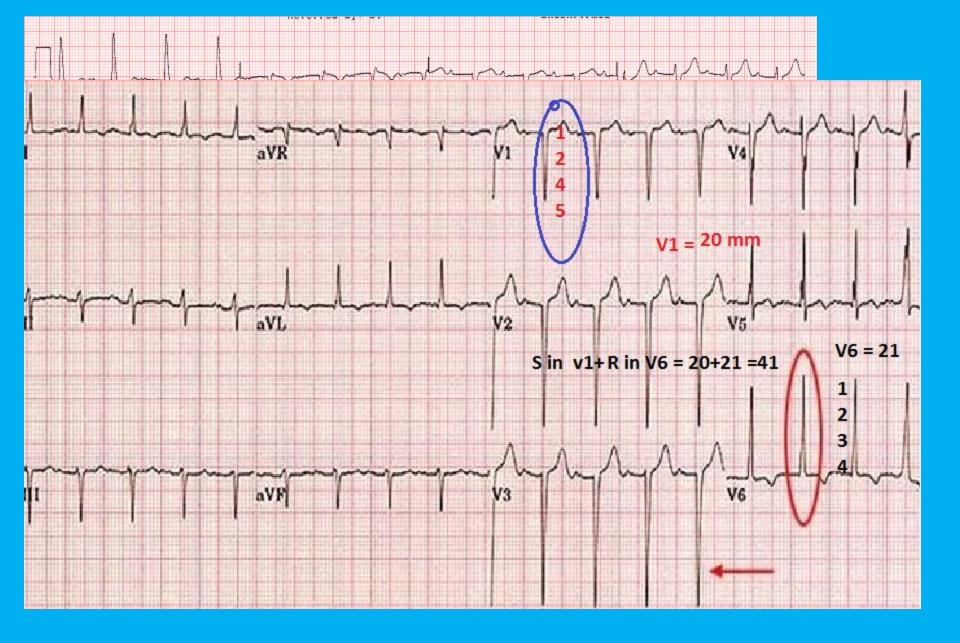


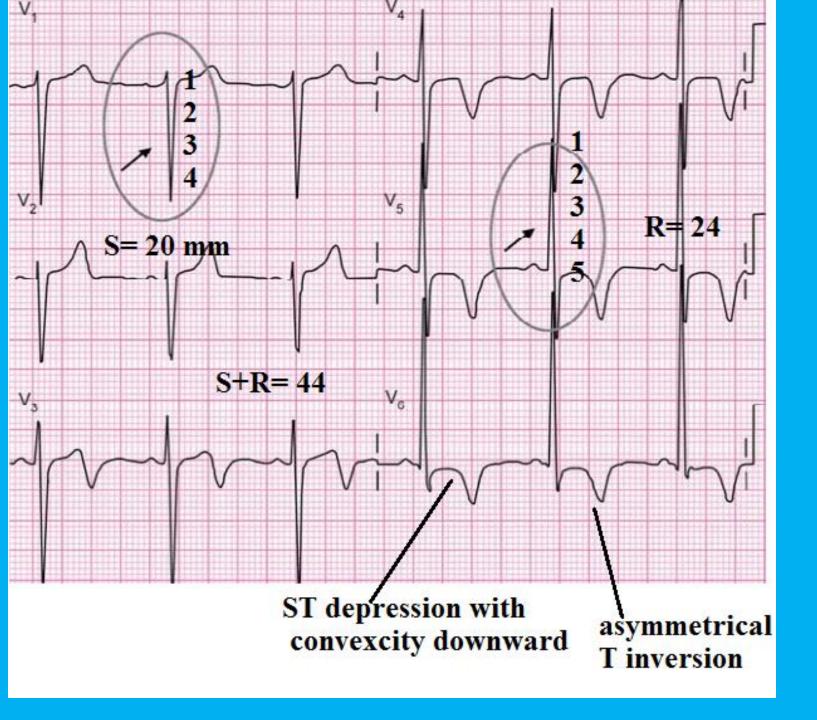


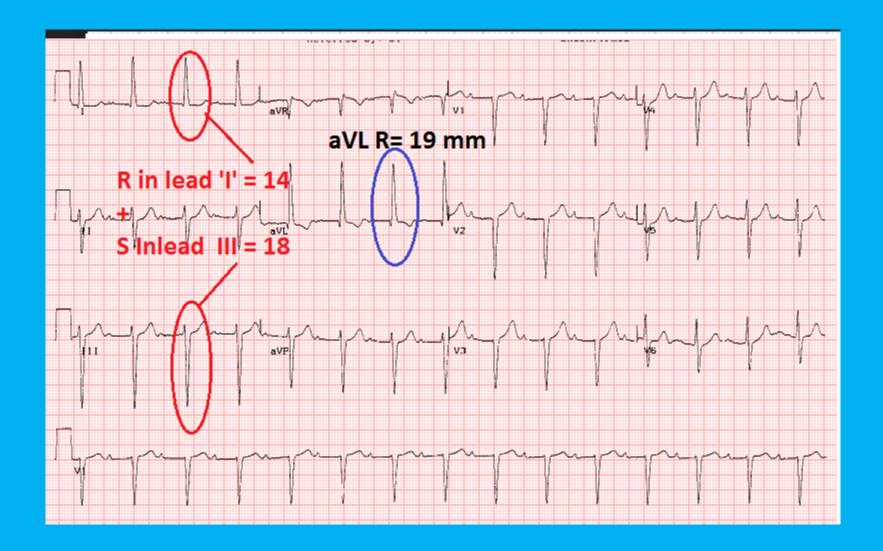
LEFT VENTRICULAR HYPER TROPHY WITH STRAIN

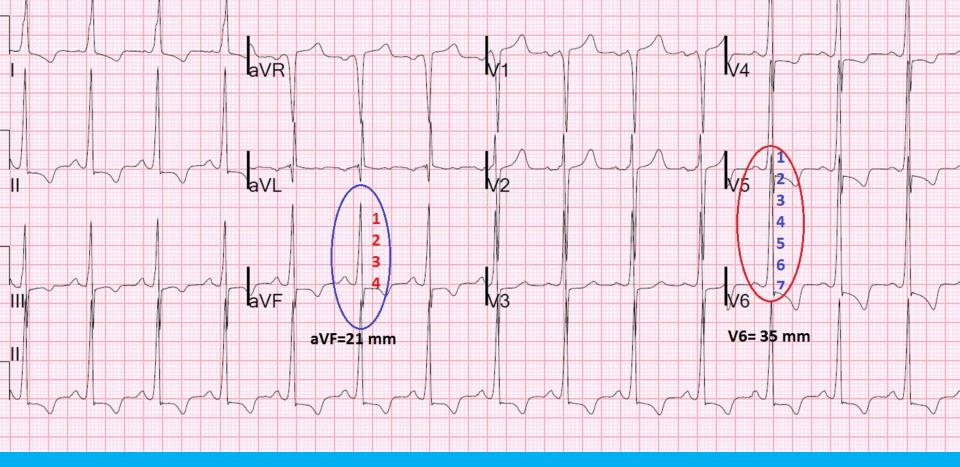


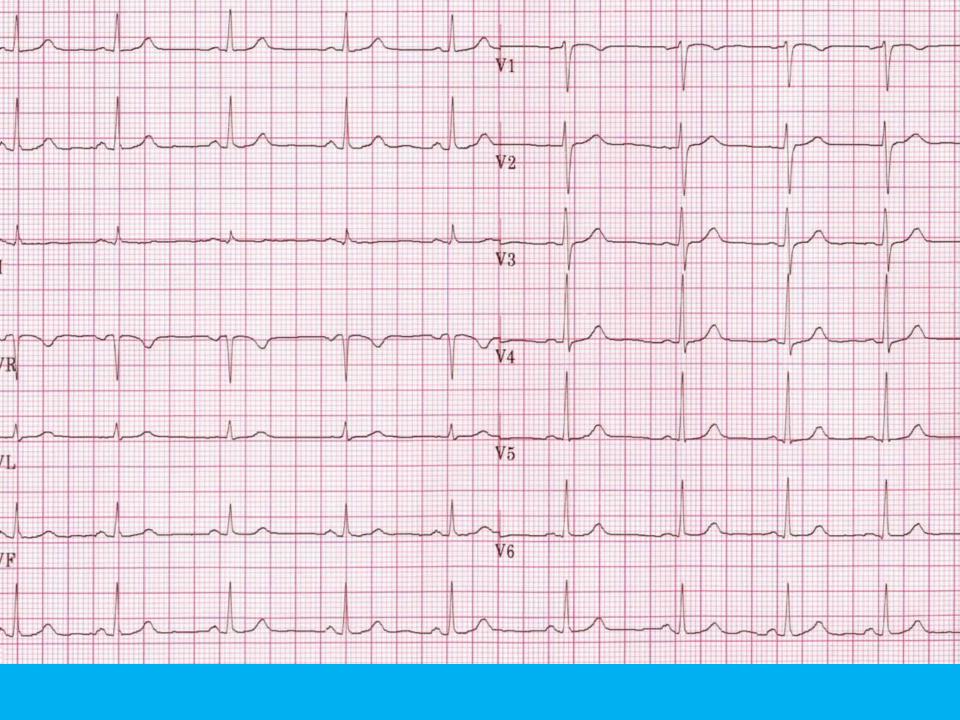


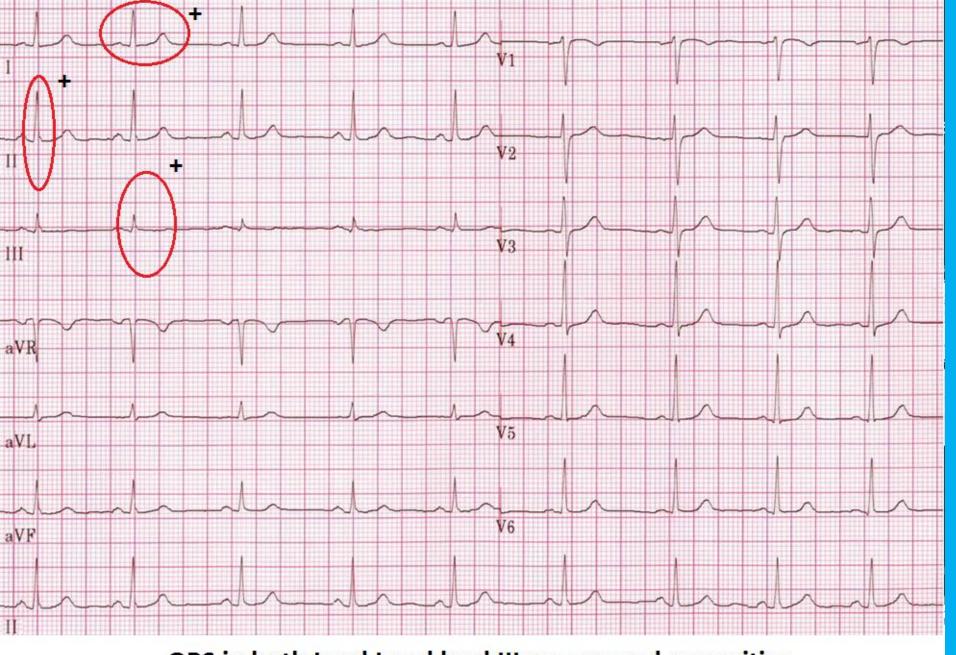




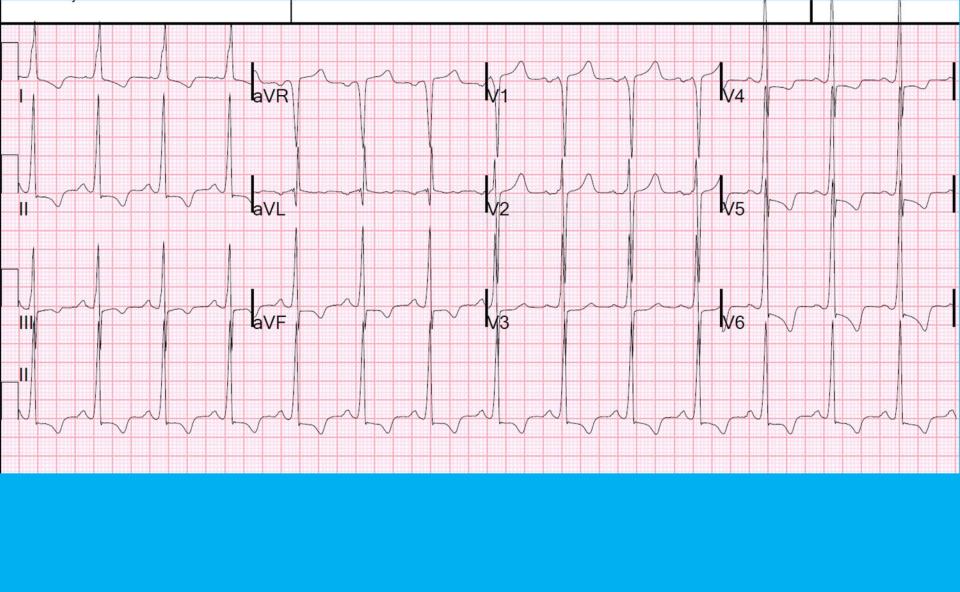


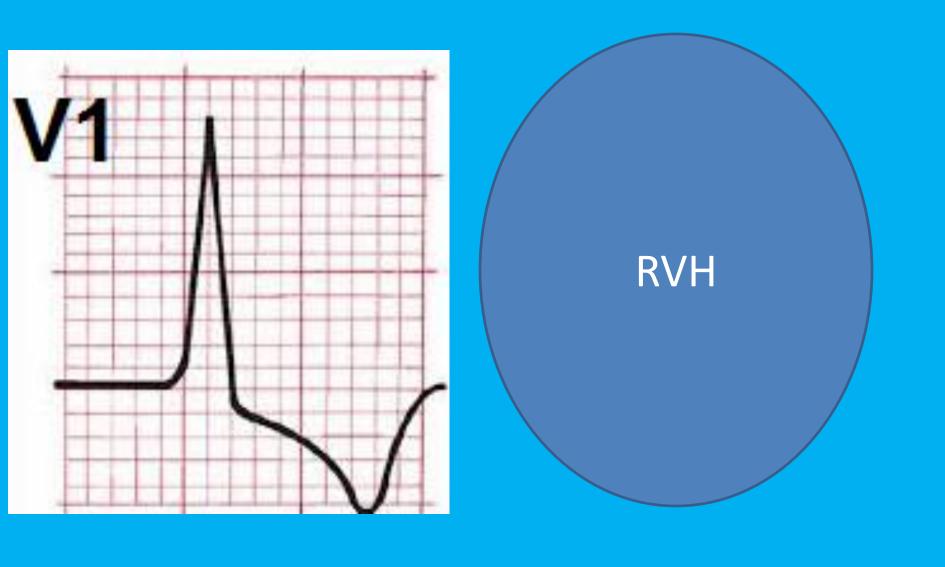






QRS in both Lead I and lead III are upward or positive normal axis





Right ventricular hypertrophy (RVH)

Right ventricular hypertrophy (RVH) If full field of the following

R in $V_1 > 7$ mm

R/S ratio in $V_1 > 1$ (R > S in V_1)

In the summation of R in V1 + S in V6 > 11

if RVH with T inversion or ST depression (V 1 to V 2)= RVH with strain

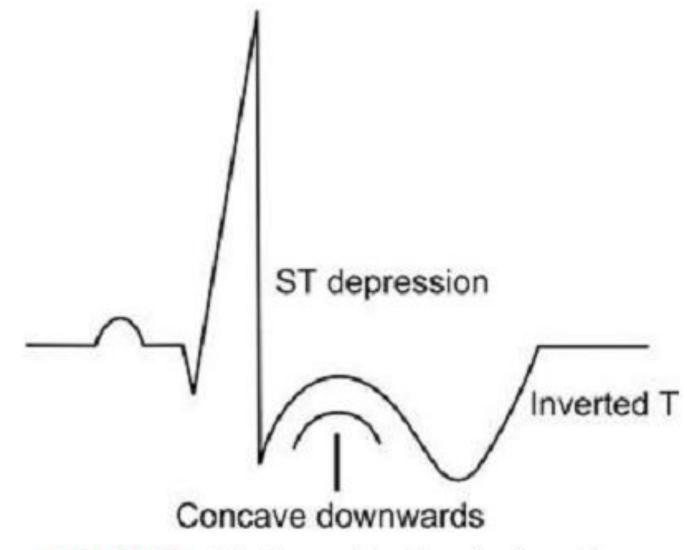
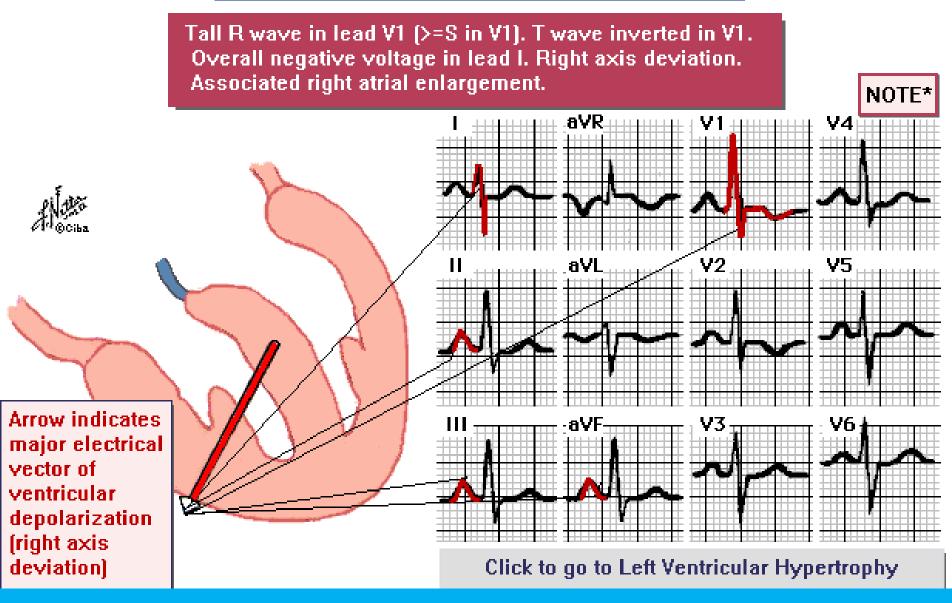


Fig. 7.40: Right ventricular strain pattern

ST depression with concavity downward & asymmetrical T inversion

Right Ventricular Hypertrophy (RVH)

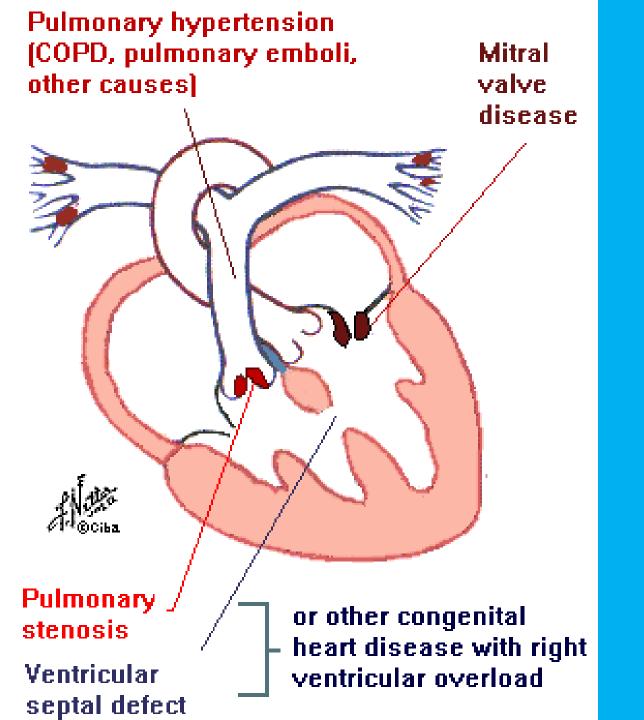


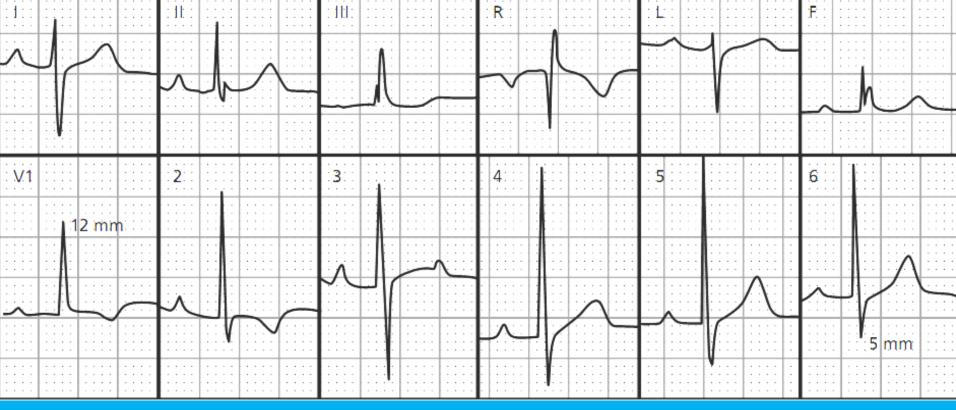
Cause of RVH

- 1.Cor-pulmonale
- 2.Pulmonary HTN
- 3.MS with pulmonary HTN
- 4.ASD
- 5.VSD
- 6.TR and Fallot's tetralogy

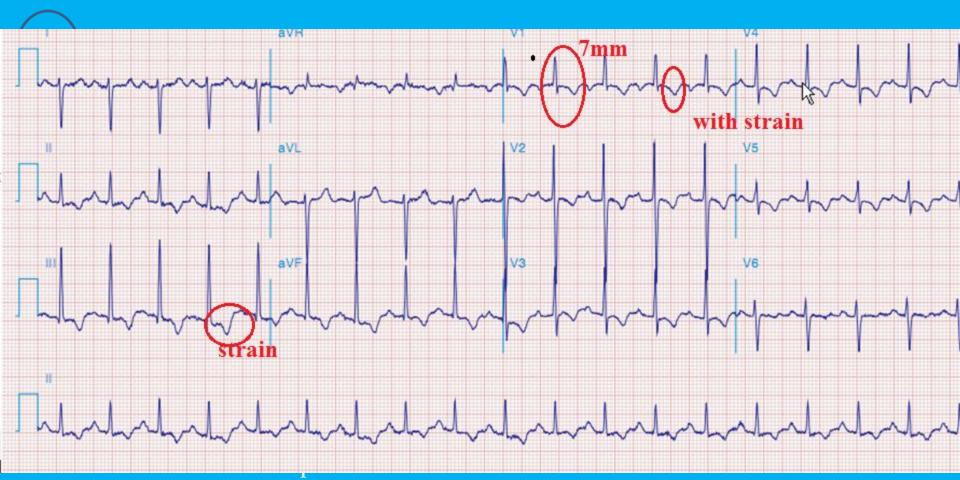
Clinically

- Left parasternal heave and
- Epigastric pulsation

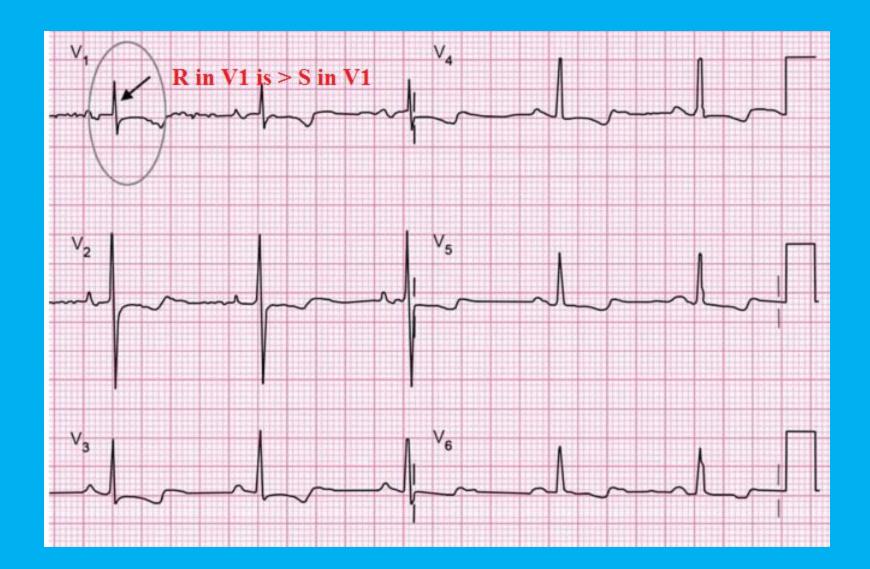


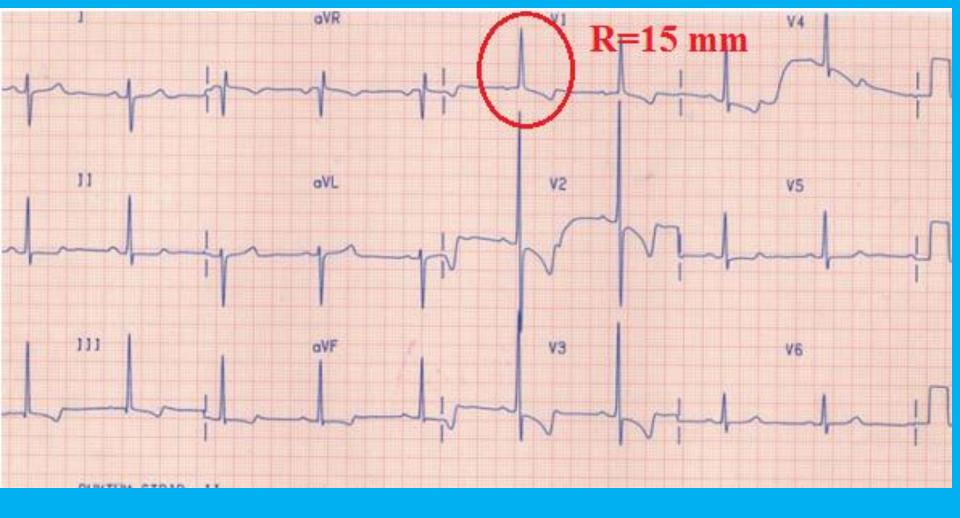


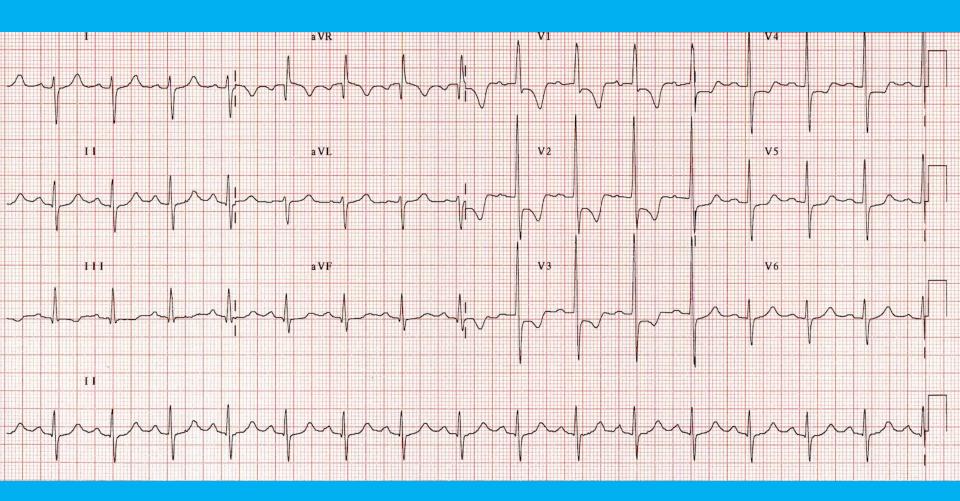
Right ventricular hypertrophy (RVH)

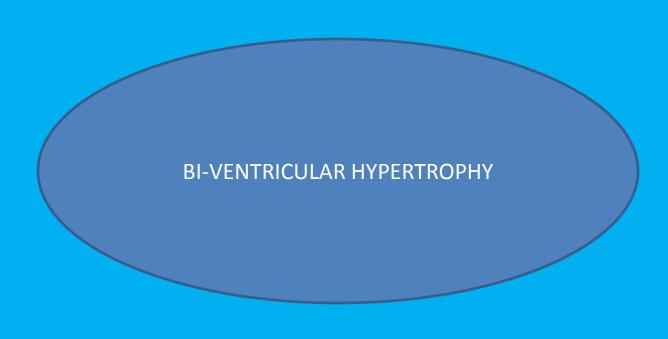


❖ p -pulmonale









Biventricular failure If ECG shows

ULVH + RVH OR **Biventricular failure**

- •Esenmenger syndrome
- Cardiomyopathy
- **OMultiple valvular disease**

■ LVH + right axis deviation

OR

 \square LVH + R> S in V₁

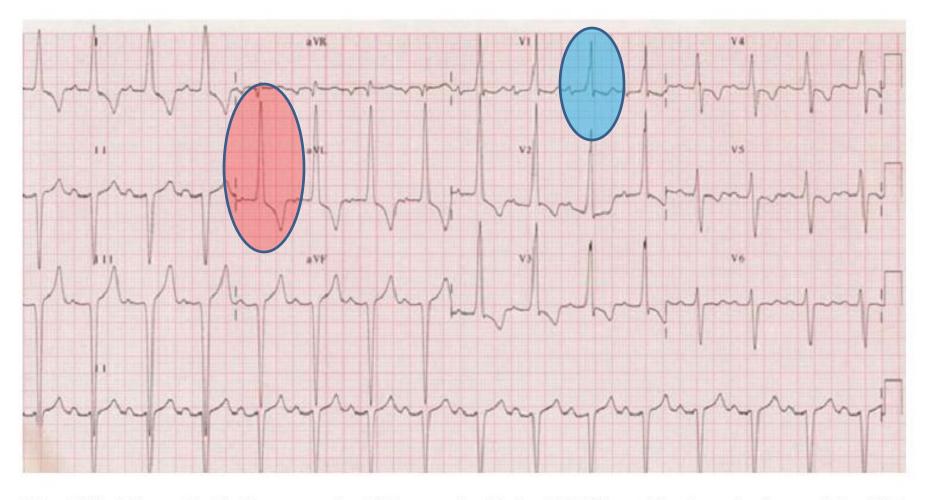
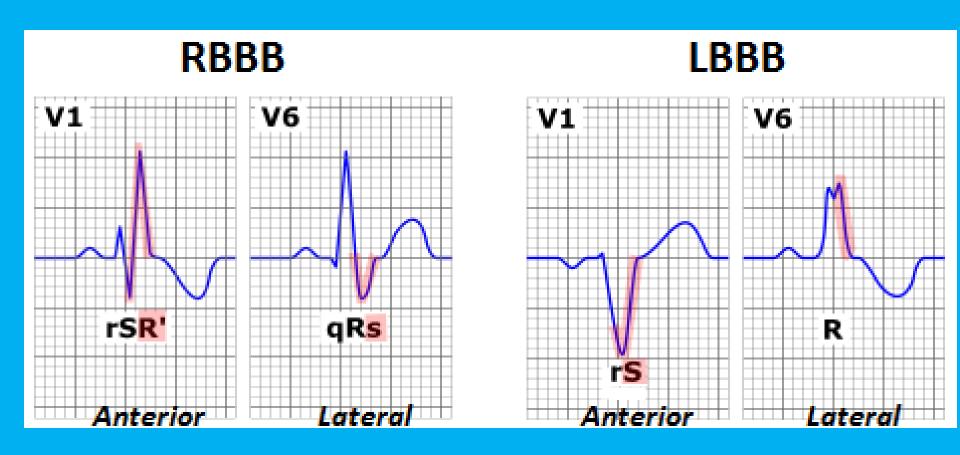
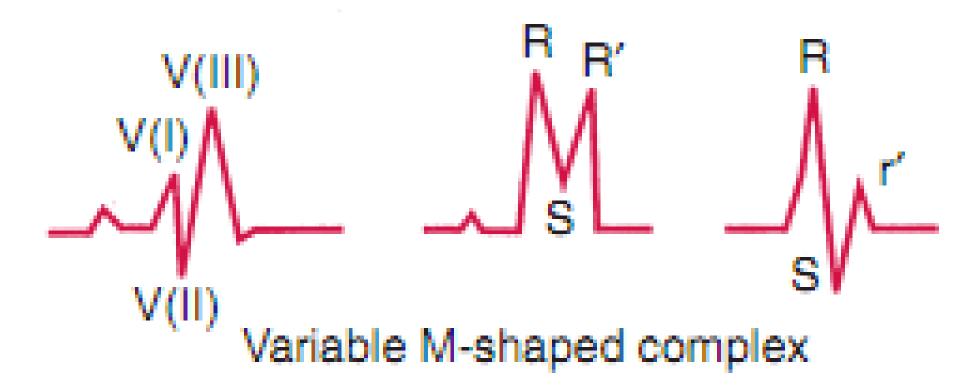


Fig. 5.14 Biventricular hypertrophy. Voltage criteria for LVH found in frontal plane with tall R waves in lead V1



RBBB

Electrode V₁

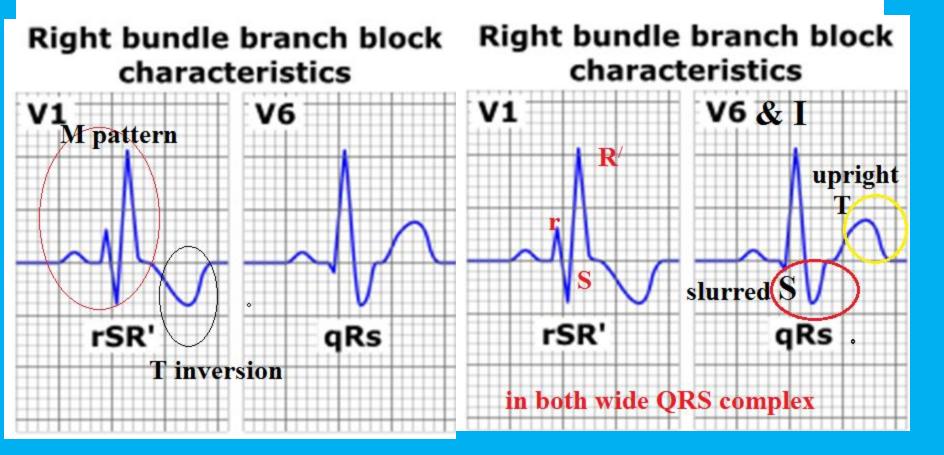


Clue to diagnosis is M in V1 or and V2

Right Bundle Branch Block

Criteria

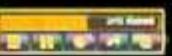
- i. Wide QRS complex
- ii. RSR pattern or rabbit ear pattern in V1
- iii. Broad and slurred S wave in leads I and V6
- iv. Right axis deviation may be present Slurred S wave in leads I and V6 are the major criteria that have to be looked.

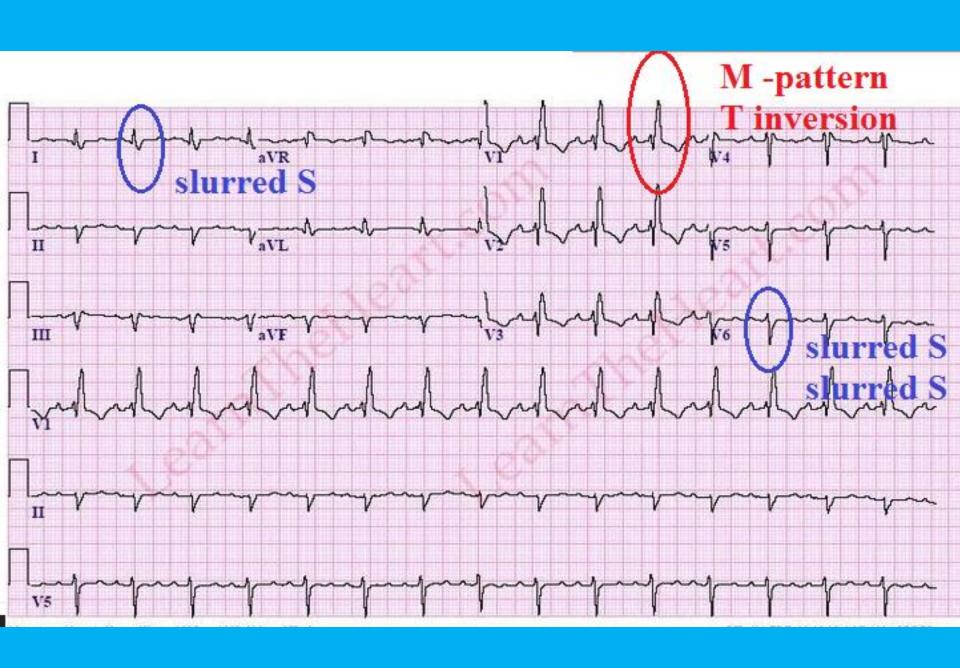


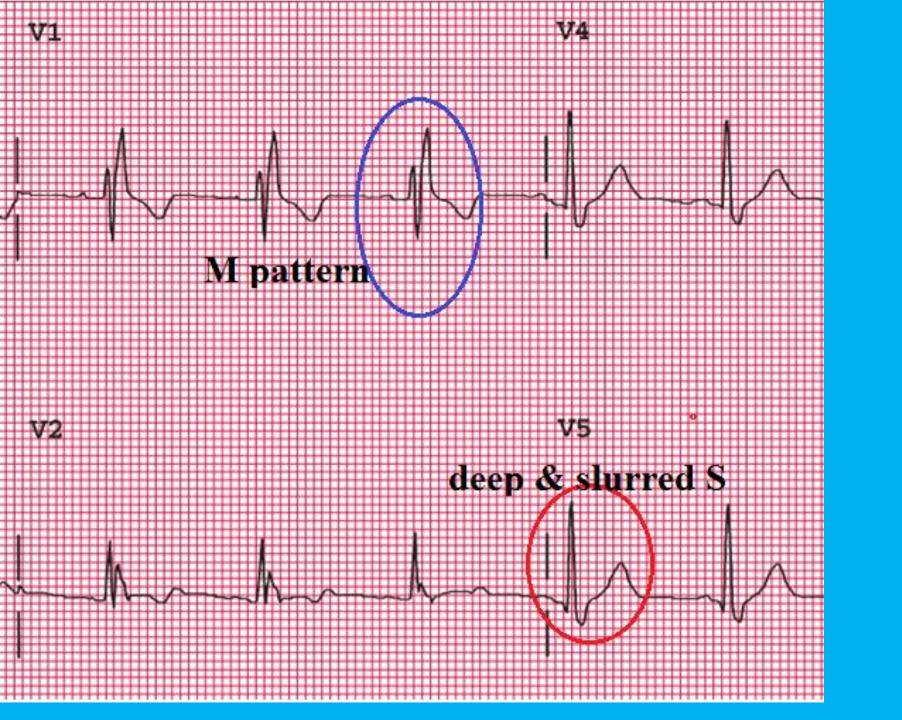
Right Bundle Branch Block (wide QRS)

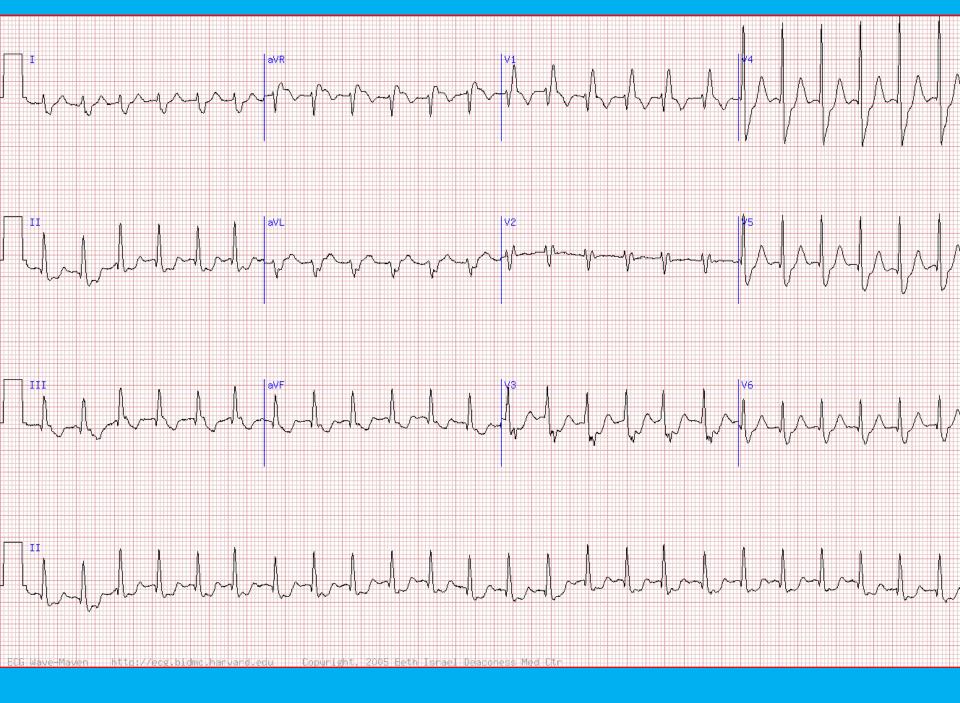
V1 will show an rSR' (R' = R prime) pattern V1 will be positively deflected - not normal

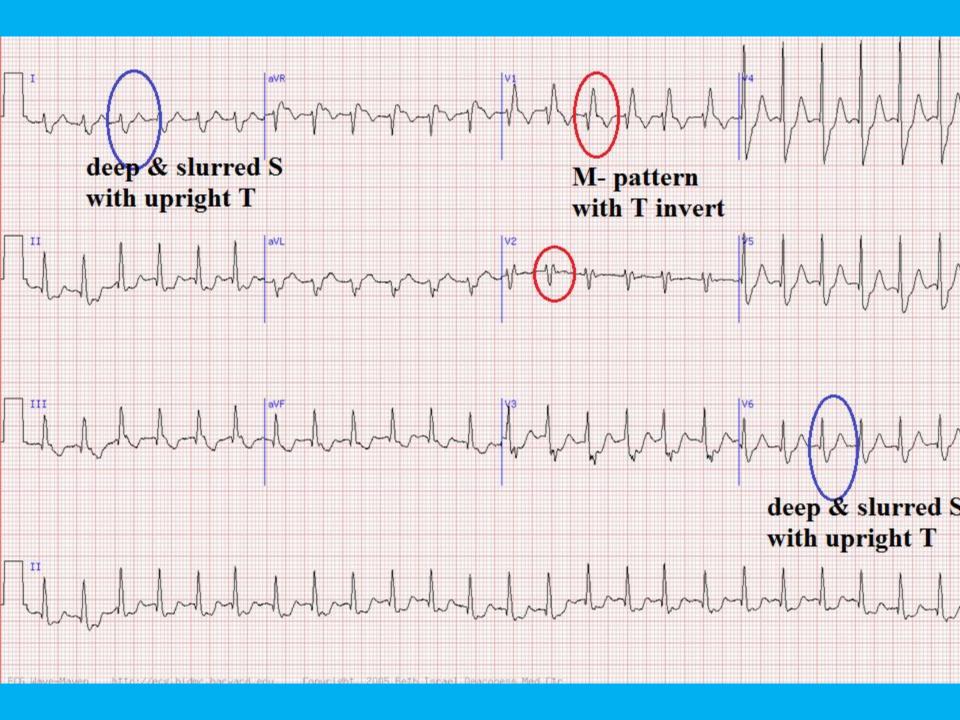


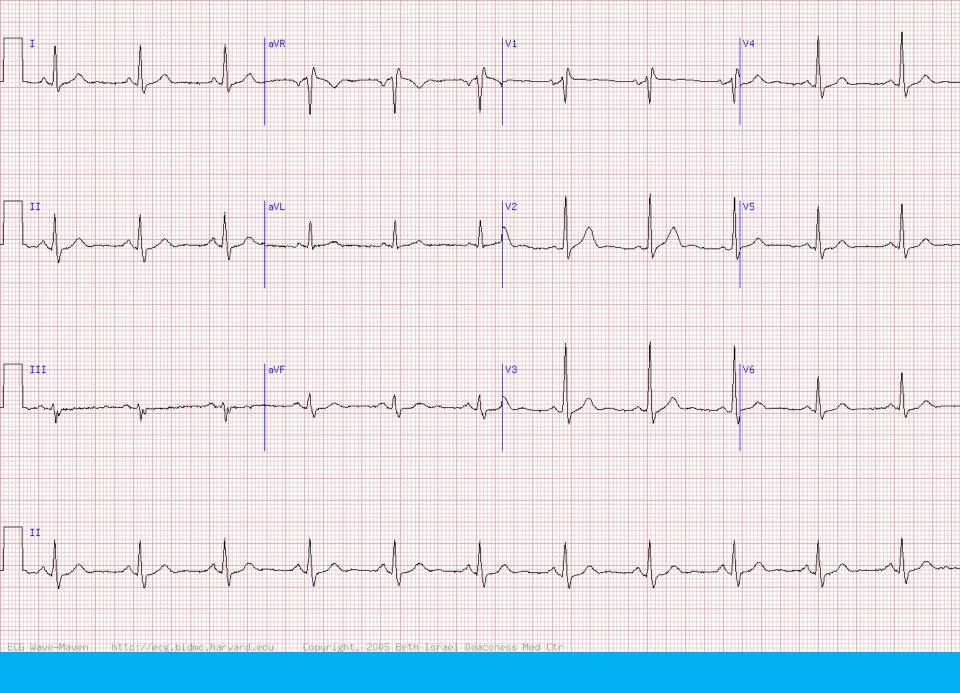


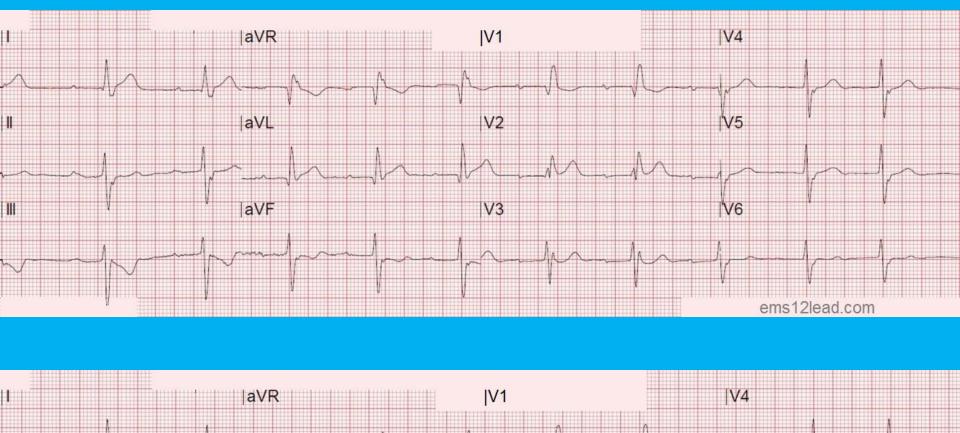


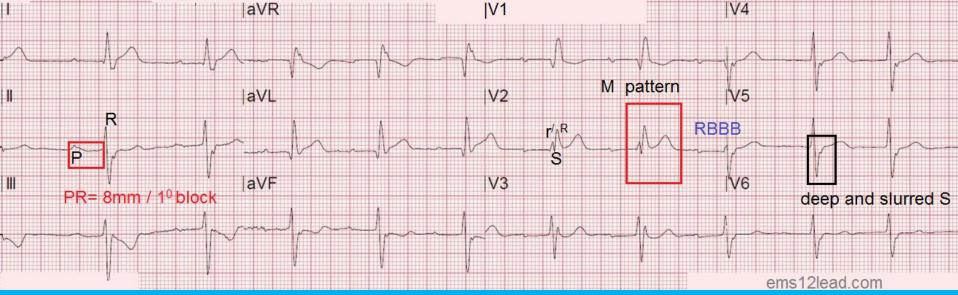






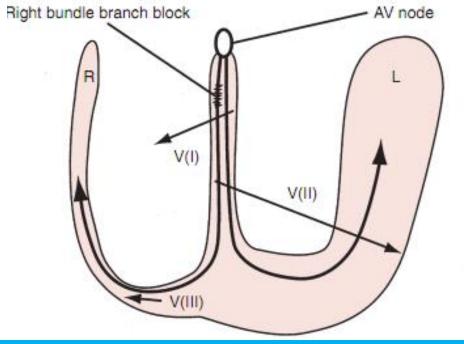






For those who r interested to know why M pattern developed in RBBB

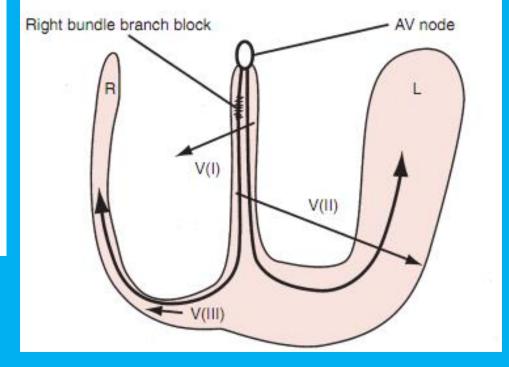




Step. One:

- ☐ We know Septum is depolarized by from left to right by LBB
- □ Vector direction toward the right that means toward V1 and away from the V6.
- ☐ That why it cause positive wave (R) in V1 and negative(Q) in the lead V6

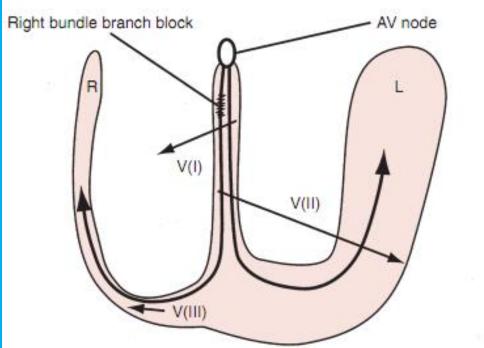


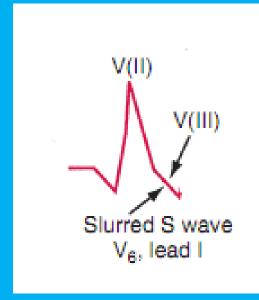




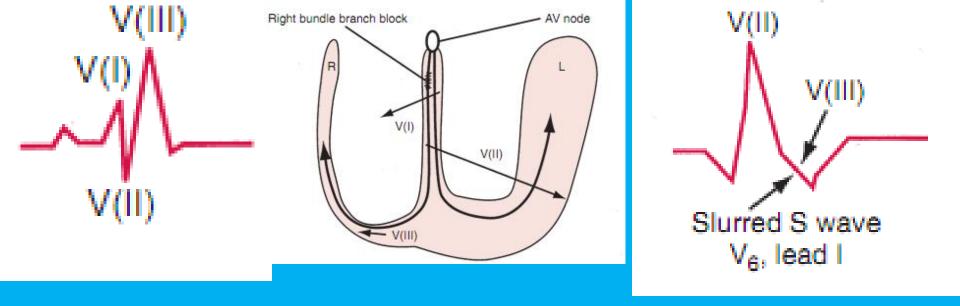
- ❖ Next left ventricle will depolarized by LBB so vector is directed towards left side.
- ❖ That means toward V6 and away fromV1.
- ❖ So it create positive wave (R) in V6 and negative wave (S)in V1



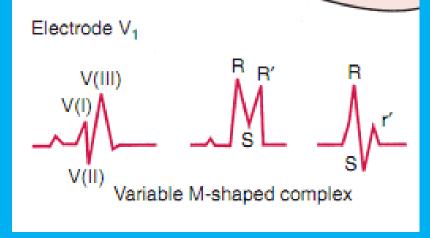




Now it is time for activation or depolarization of righty ventricle .It is done by RBB but here RBBB is blocked that whys it dose not act .

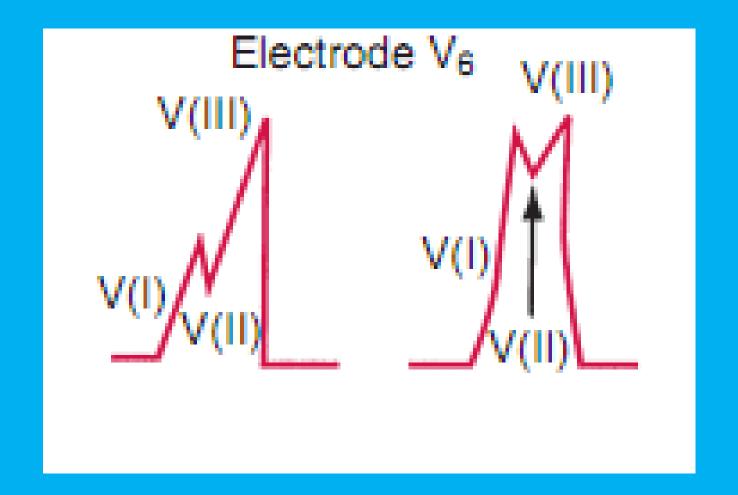


- ➤ After depolarizing LV, LBB now will activate or depolarize d right ventricle.
- ➤ Net effect is vector directed toward the right so it create another positive wave (R) in V1 and as its direction is away from V6 it cause s negative wave(S) in the lead V6



- **□**Wide QRS ≥0.12 second
- \square A secondary R wave (R') in V₁ or V₂ (i.e., an rSR', rsR', or rsr' complex hat often is M-shaped).
- □ The secondary R wave (R') is usually taller than he initial R wave
- \square A wide, slurred S wave in leads V₅, V₆, and I with duration >40 ms;
- □the S wave is longer in duration (length) than the preceding R wave in leads
- ■V6 and I
- ☐ The axis may be normal, right, or left. If left axis is present, consider left anterior fascicular block (hemiblock) (see Chapter 9).

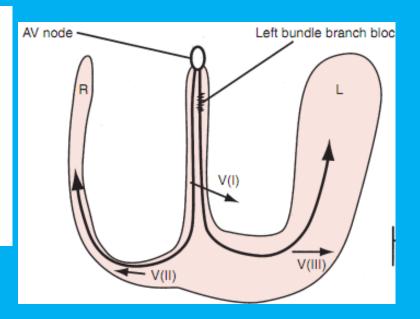
LBBB



Clue to diagnosis is M in V5 or and V6

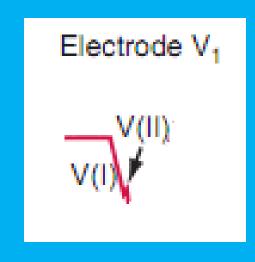
Electrode V₁

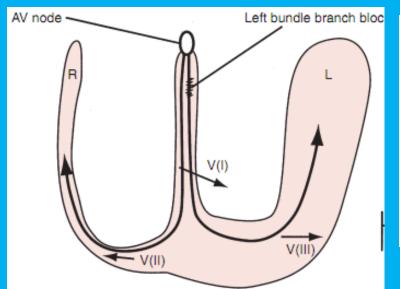






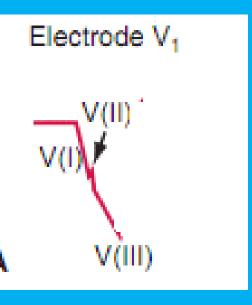
- □We know Septum is depolarized by from left to right by LBB
- □But here LBB is blocked sot it will be depolarized by the RBB from right to left .
- \square As the Vector direction toward the left that means away from V1 and toward the V6 .
- □That why it cause negative(Q) in V1 and positive wave (R) in the lead V6

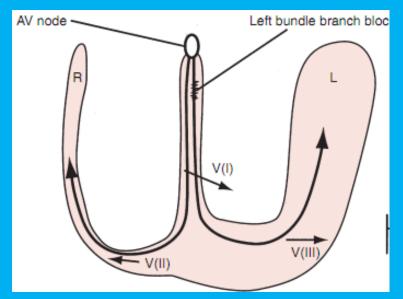


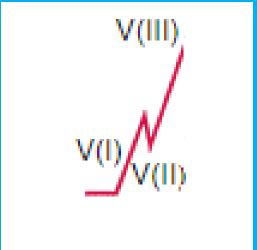




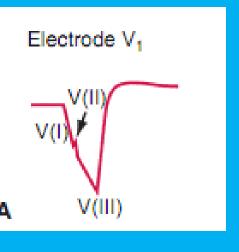
- Now as depolarization wave / Vector II travels from left to right through the right ventricular mass and depolarize the right ventricle.
- ❖It cause a upward notch in the Q wave of V1 (as depolarization toward the lead) &
- *Causes a downward notch in the R wave of V₅ (as depolarization oposite the lead)

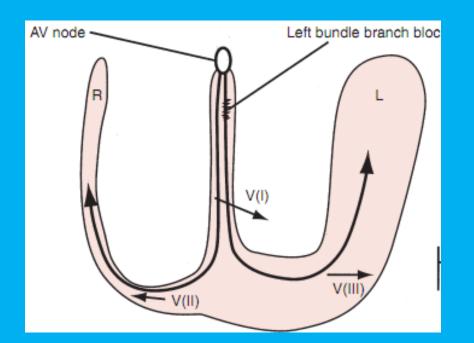






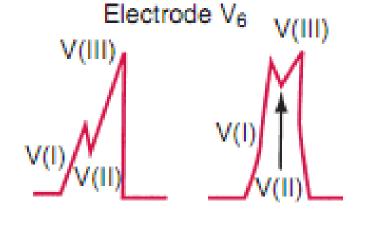
- ❖Now vector depolarize the left right to left .
- ❖ It create further downward sloping in lead V1 and
- **❖**Upward or positive wave in V 6 which appear as M .







The marked derangement in depolarization of the left ventricle causes the ST segment in leads V1 through V4 to be abnormally elevated. In presence of LBBB it is to difficult to comment on MI.



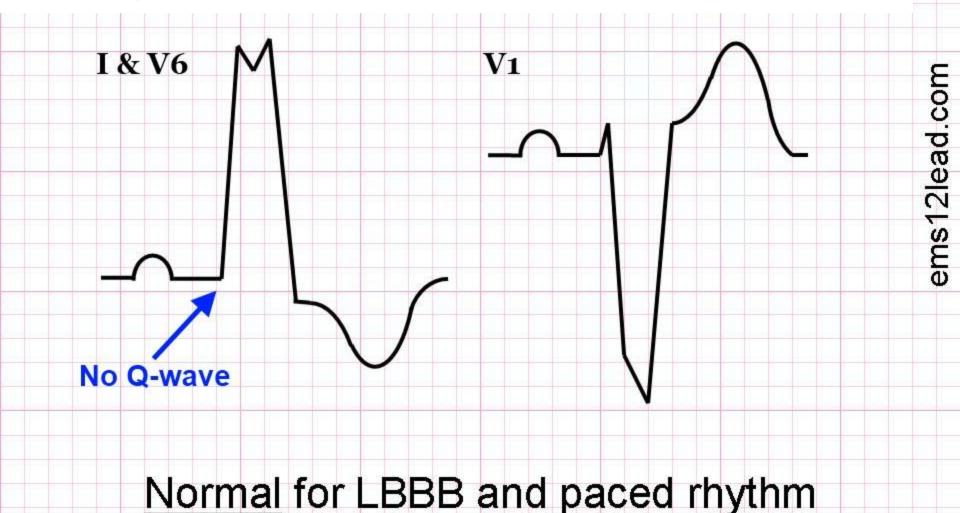
Diagnostic Criteria

- **♦** QRS duration ≥0.12 second.
- *RSR`(M pattern)/ notching in V5 or V6 or also in lead I and AVL
- **❖**Leads V1 and V2 reveal QS or rS pattern with poor R wave progression in V2 and V3

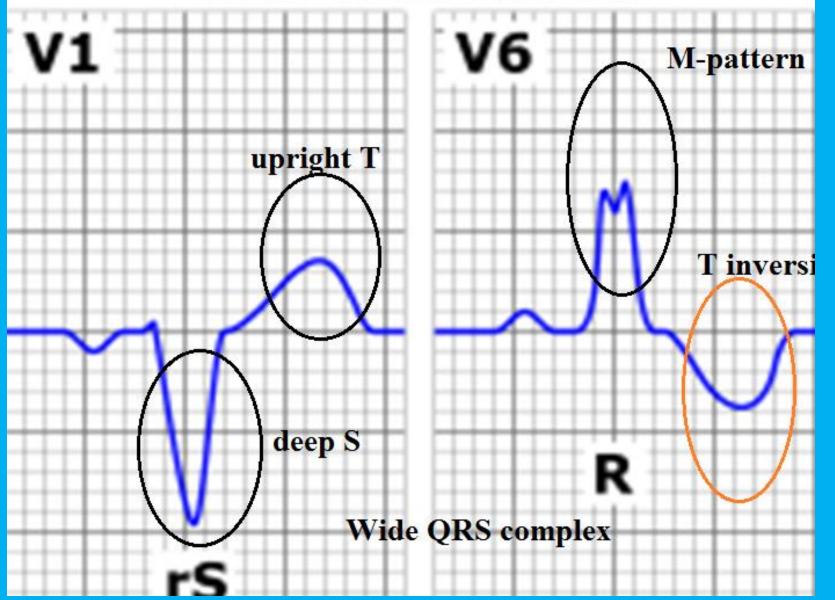
Diagnostic Criteria of incomplete LBBB may be made

•if the QRS duration is 0.10 to 0.11 second with notching of the R wave in V5 or V6

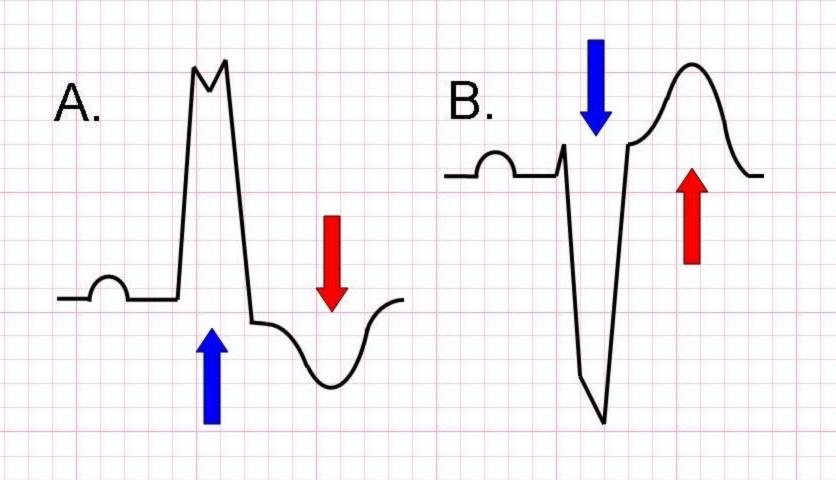
- i. Wide QRS complex with duration of > 0.12 s (> 3 mm)
- ii. Deep and broad S wave in V1 with no R wave.
- iii. Broad slurred R wave or RR' pattern without a Q wave in leads I and V6.
- iv. Always associated with left axis deviation.



Left bundle branch block characteristics

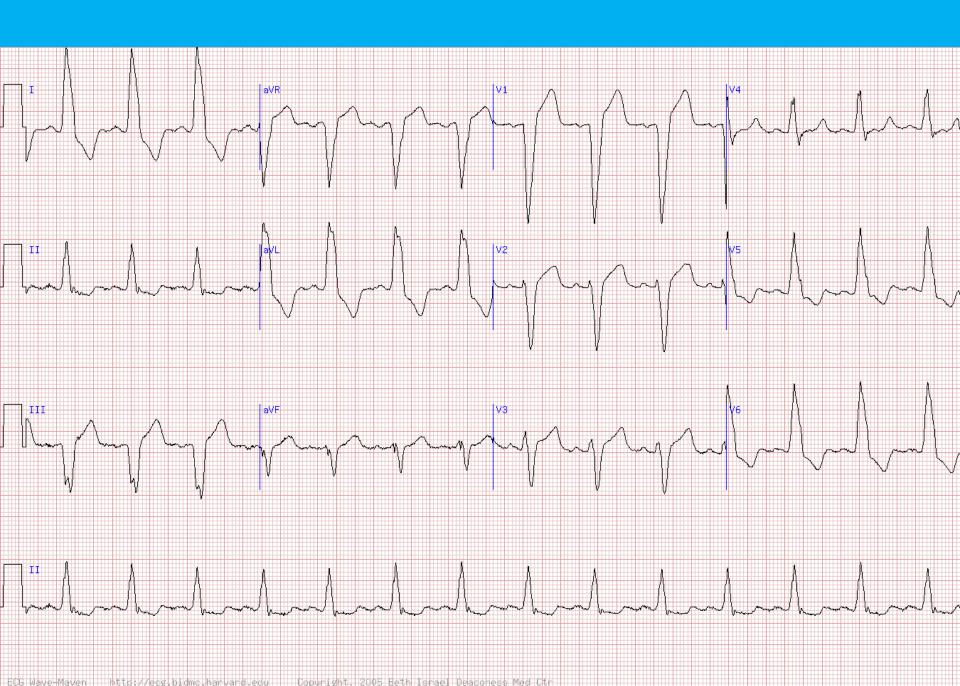


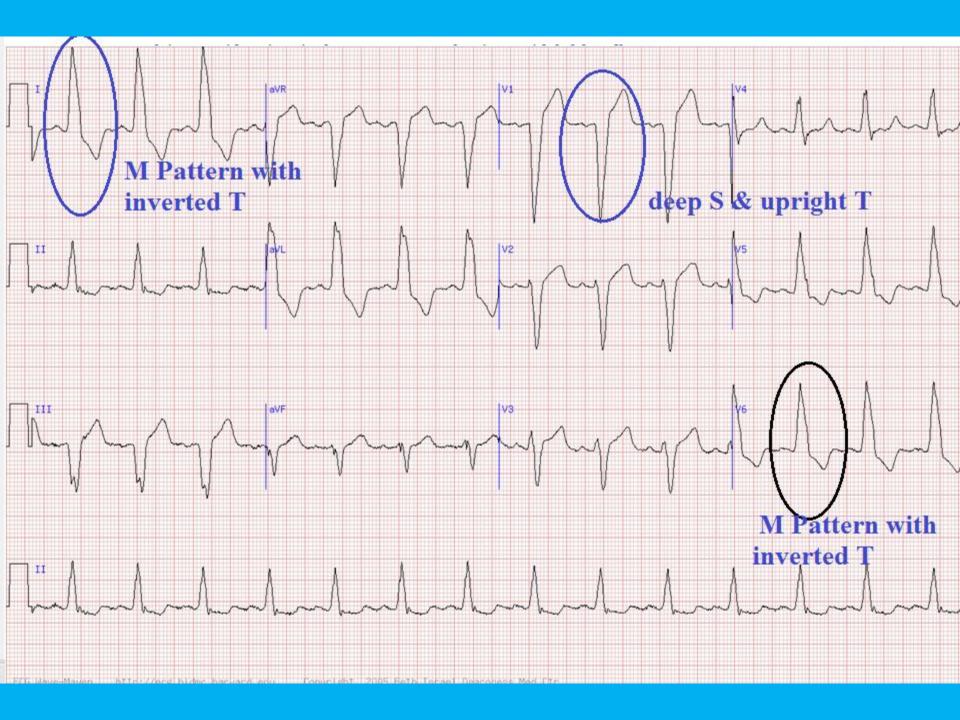
Discordant ST-Segments and T-Waves

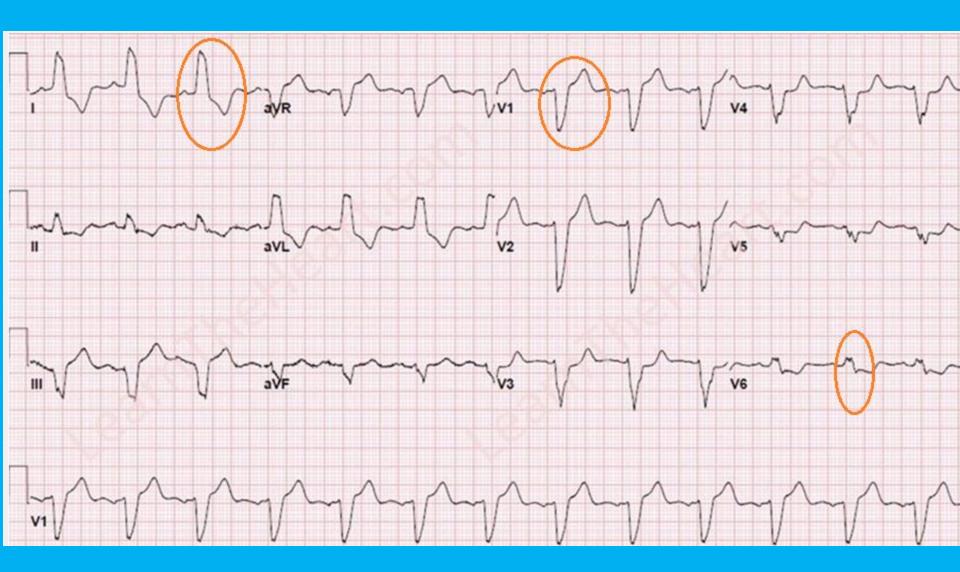


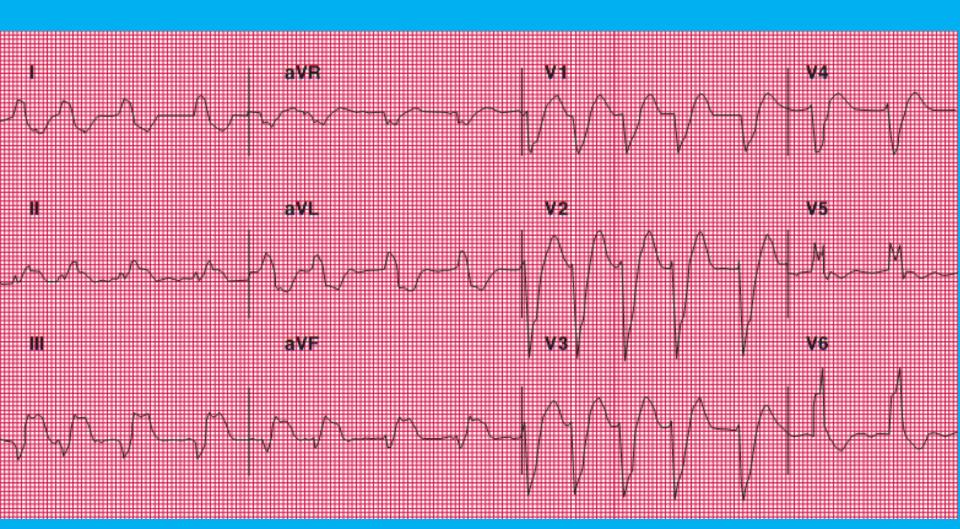
ems12lead com

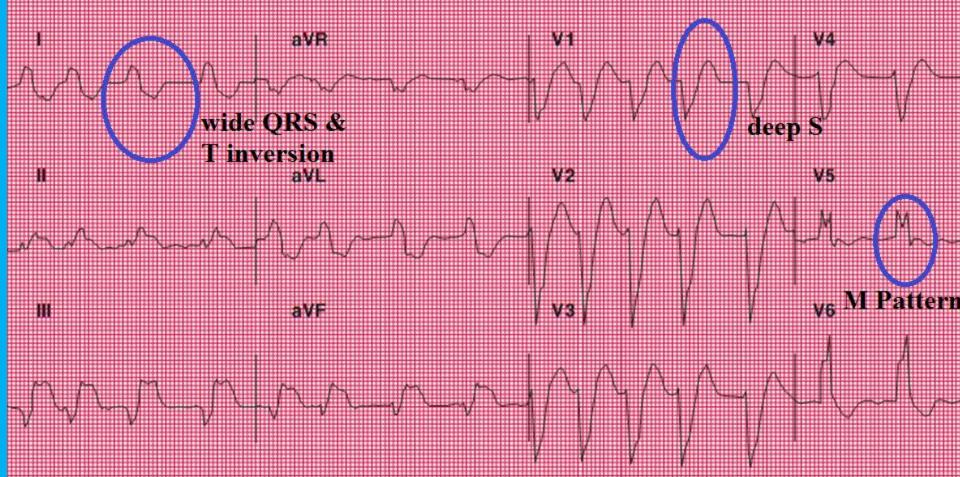
Normal for LBBB and paced rhythm

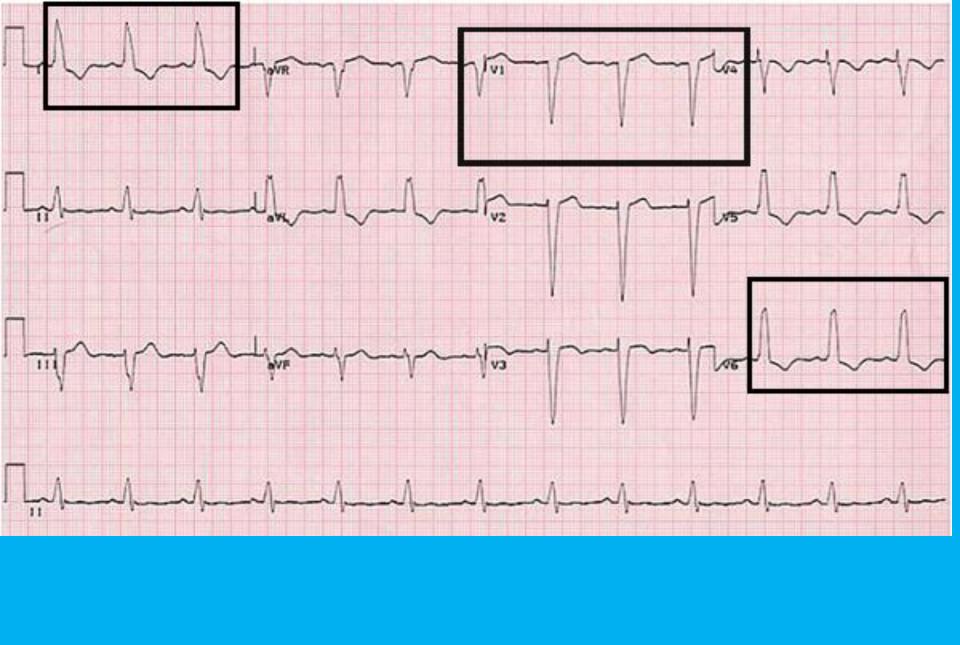


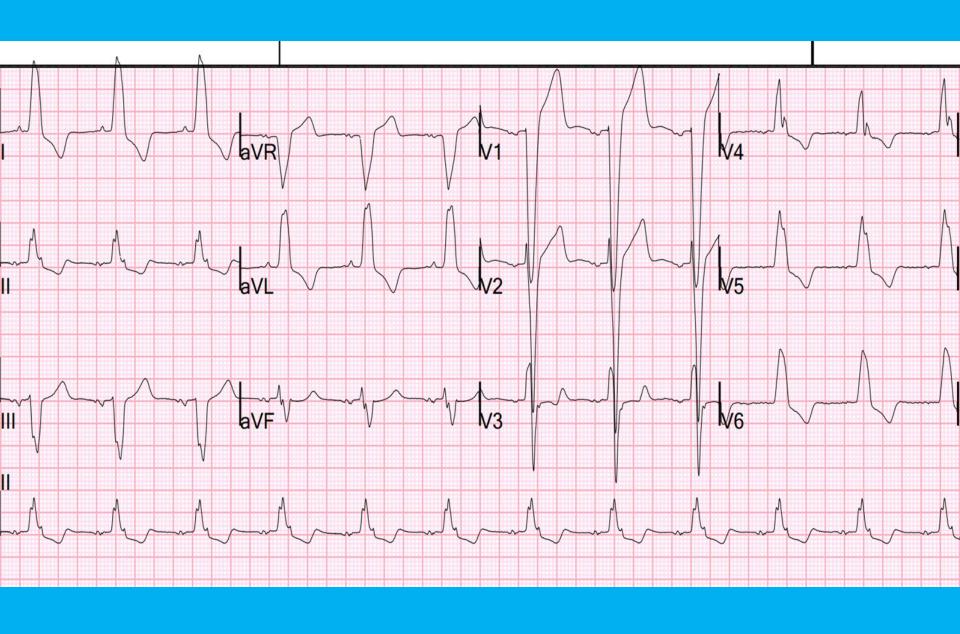


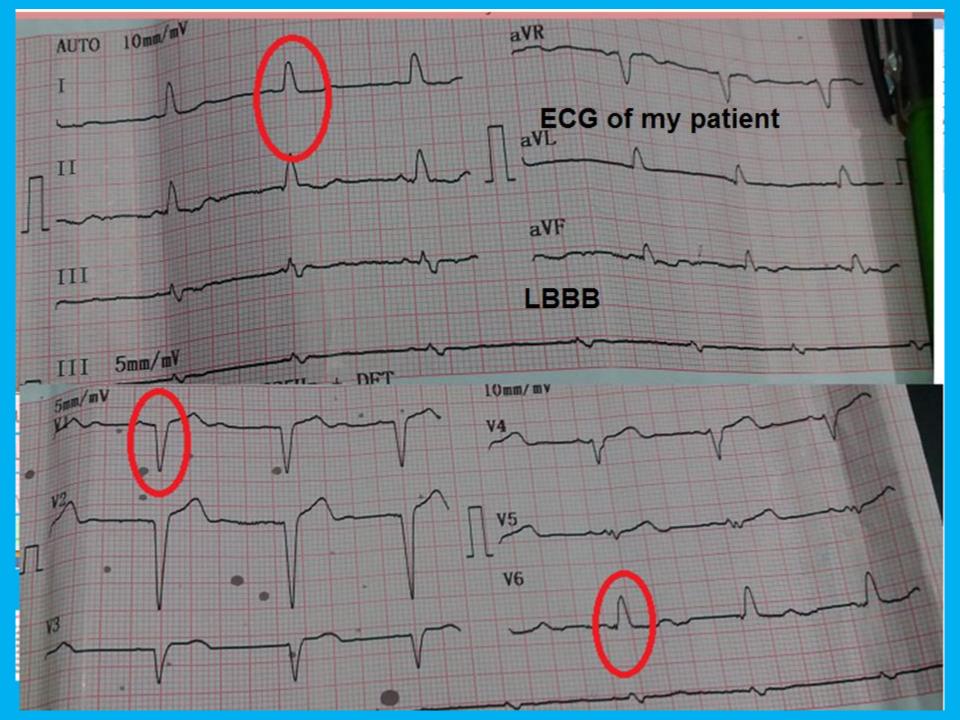










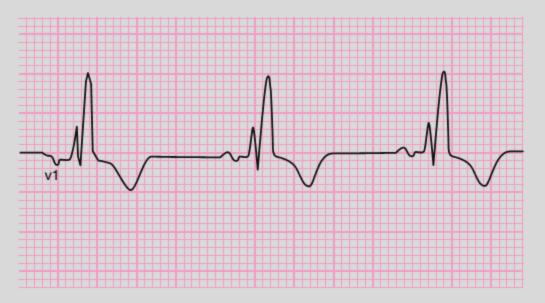


Remember

Quick way to tell right and left Bundle Branch Block apart. Widened QRS, look in lead $V_{\scriptscriptstyle \rm L}$

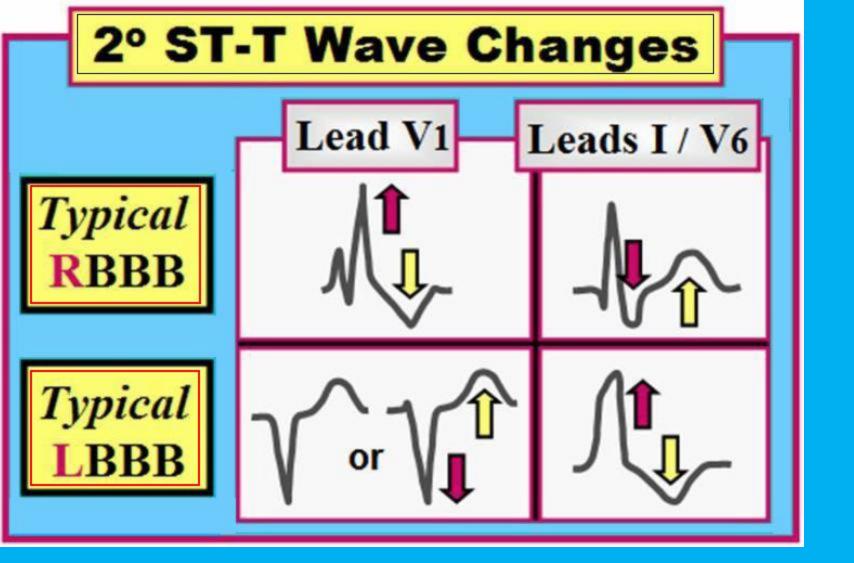
Causes

Positively deflected=RBBB

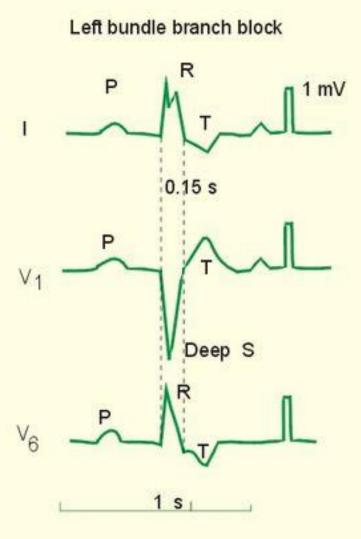


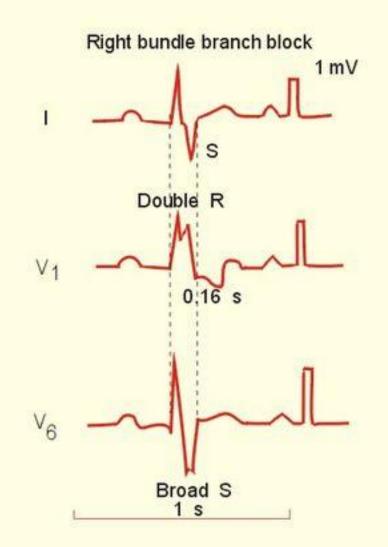
Negatively deflected=LBBB



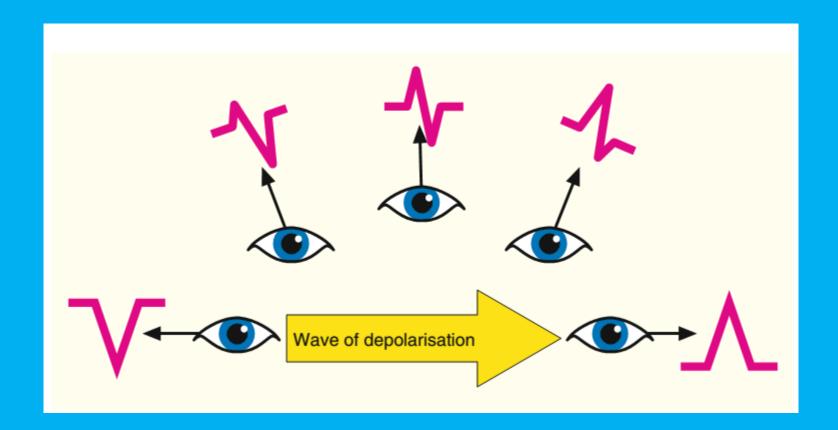


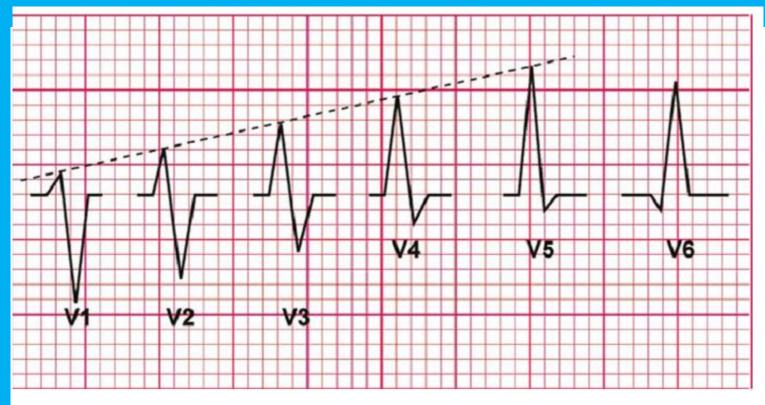
Bundle Branch Block



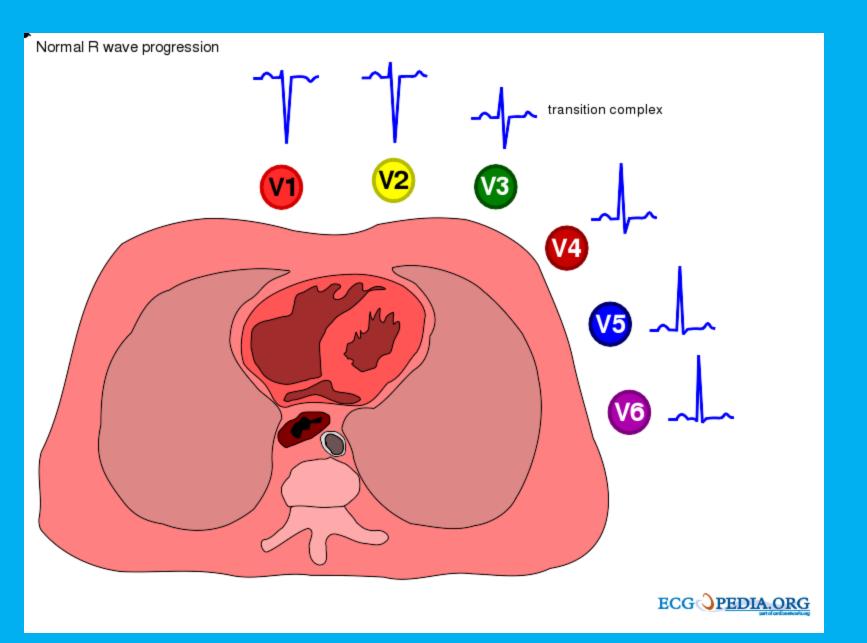


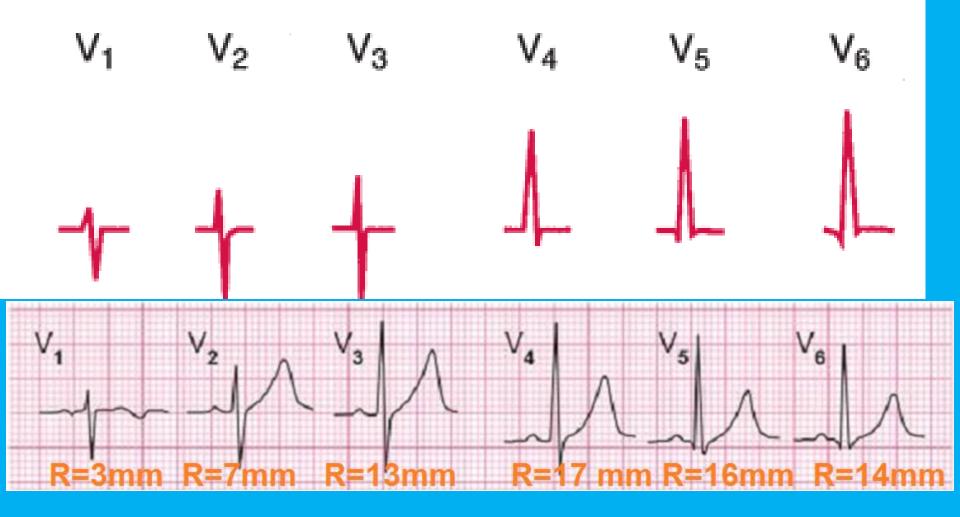
SLOW PROGRESSION OF R WAVE



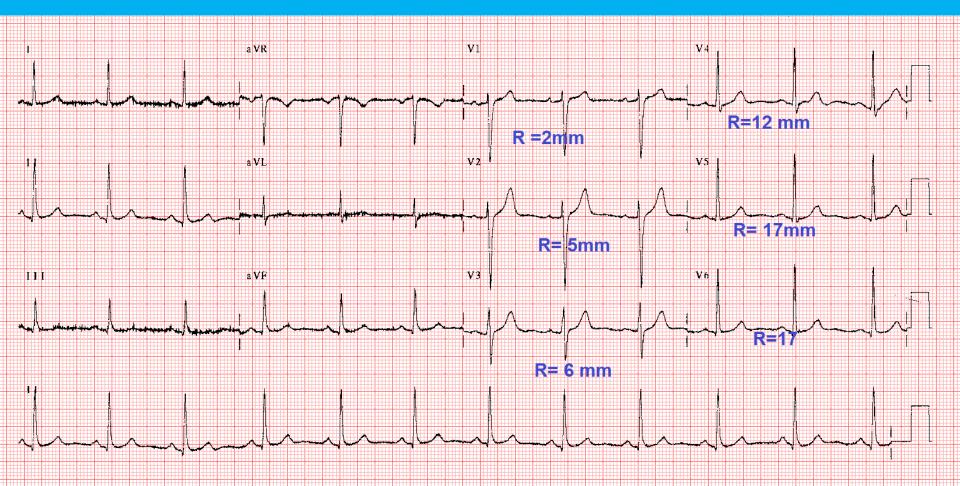


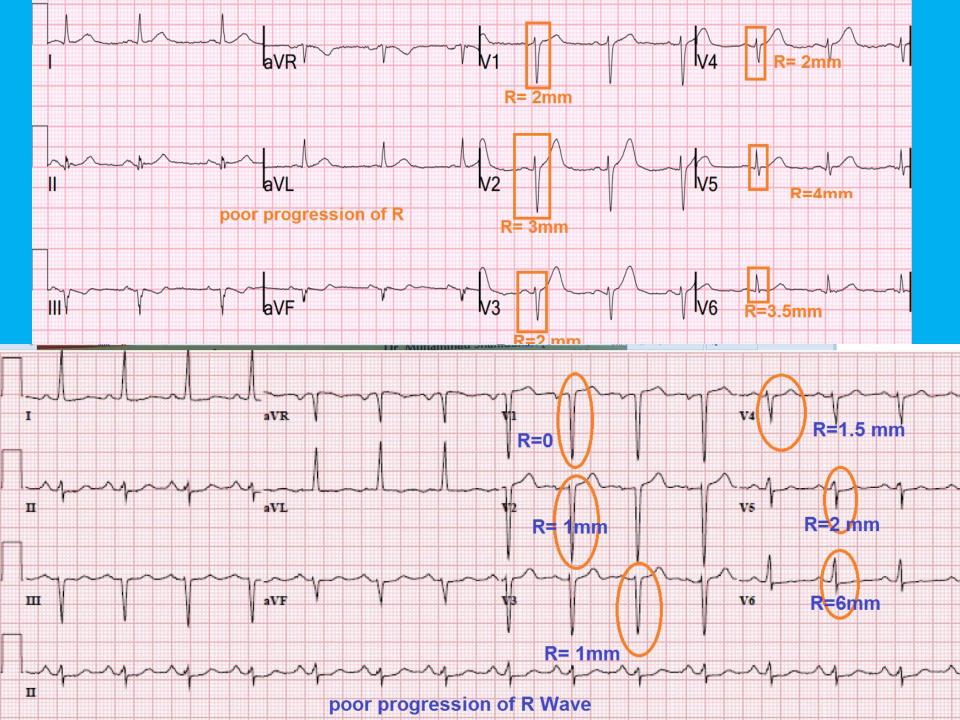
height of R is progressively increase from V1 to V6

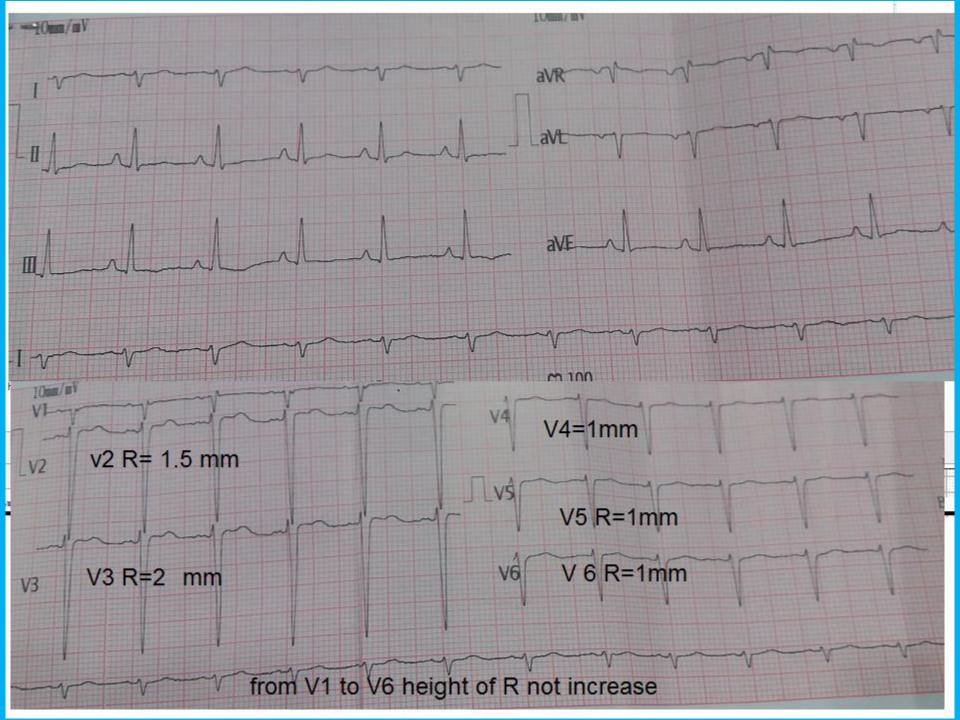


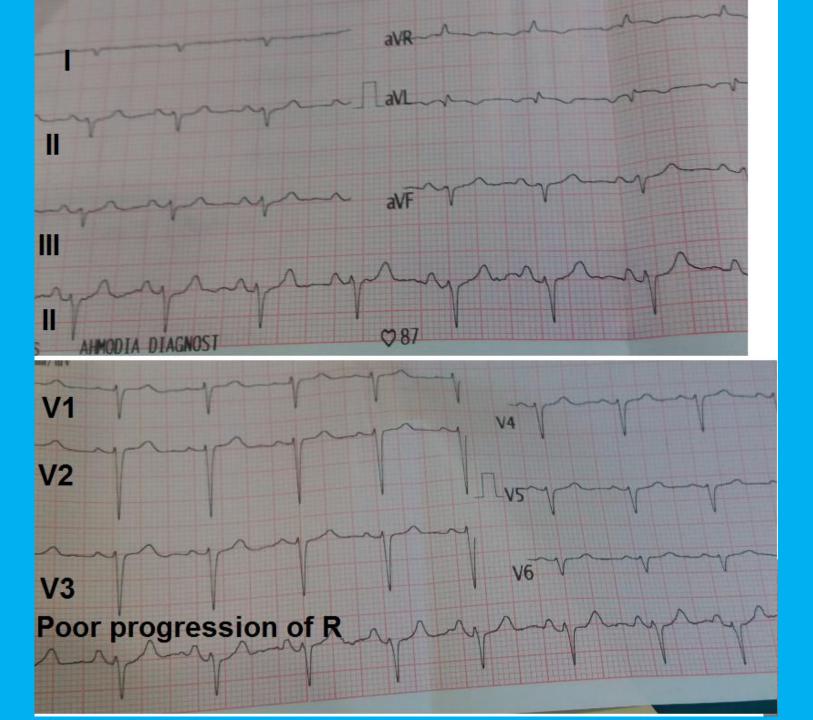


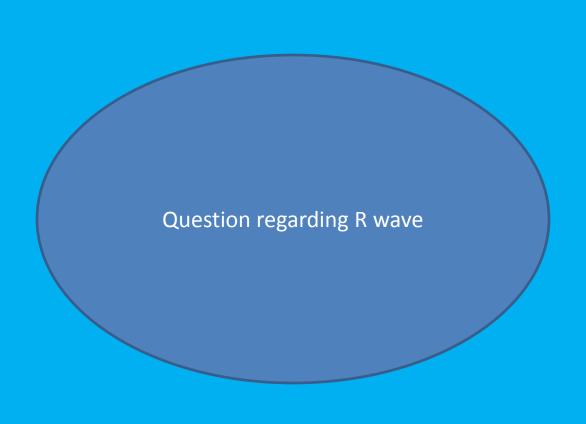
Progressively increasing the height of the R wave from V1 to V6











Tall R wave in V1:

- 1. Right ventricular hypertrophy
- 2. True posterior MI
- 3. WPW syndrome
- 4. RBBB
- 5. Dextrocardia

Small R wave:

- 1. Improper ECG standardization
- 2. Obesity
- 3. Emphysema
- 4. Pericardial effusion
- 5. Hypothyroidism
- 6. Hypothermia

Poor progression of R wave:

- 1. Anterior or anteroseptal MI
- 2. LBBB
- 3. Dextrocardia
- 4. Left sided massive pleural effusion
- 5. COPD
- 6. Left sided pneumothorax

High voltage QRS:

- 1. Improper standardization
- 2. Thin chest wall
- 3. Ventricular hypertrophy
- 4. WPW syndrome

Low voltage QRS (less than 5 mm in leads I, II, III and <10 mm in chest leads):

- 1. Improper standardization
- 2. Obesity or thick chest wall
- 3. Pericardial effusion
- 4. Emphysema
- 5. Chronic constrictive pericarditis
- 6. Hypothyroidism
- 7. Hypothermia

Wide QRS:

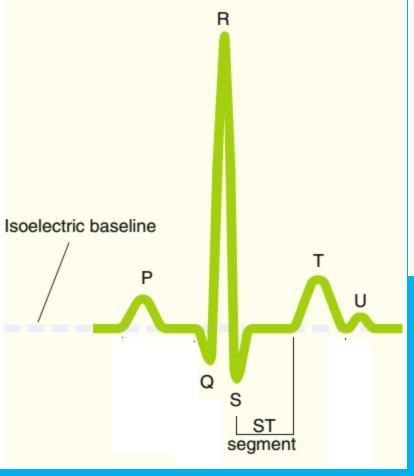
- 1. LBBB and RBBB
- 2. Ventricular ectopic
- 3. Ventricular tachycardia
- 4. Idioventricular rhythm
- 5. WPW syndrome
- 6. Hyperkalemia

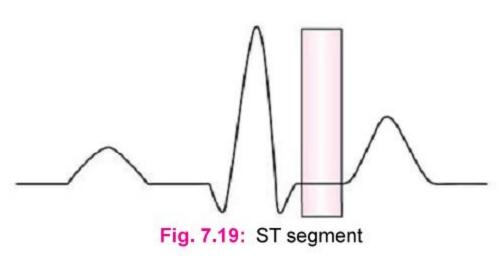
Change in shape of QRS:

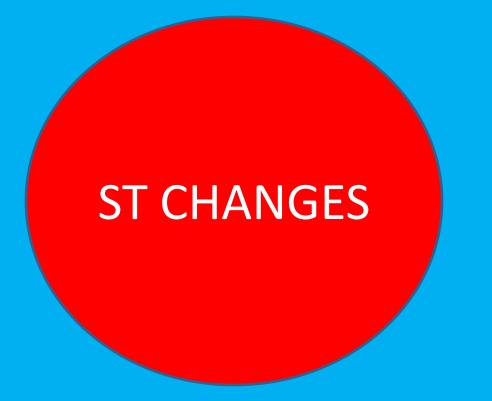
- 1. RBBB
- 2. LBBB
- 3. Ventricular tachycardia
- 4. Ventricular fibrillation
- 5. WPW syndrome

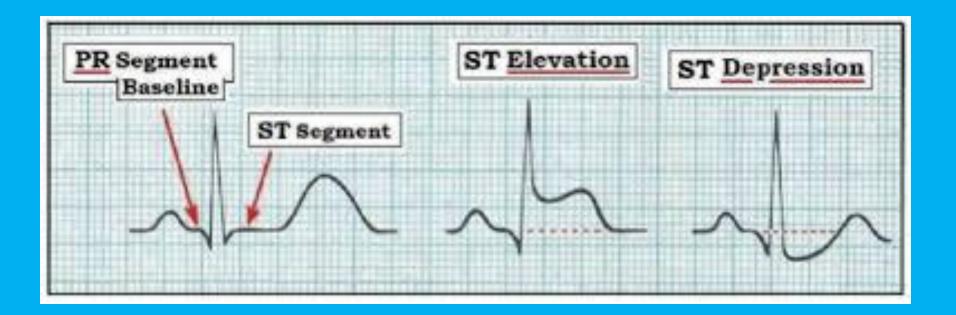
Variable QRS:

- 1. Torsades de pointes
- 2. Multifocal ventricular ectopics
- 3. Ventricular firillation

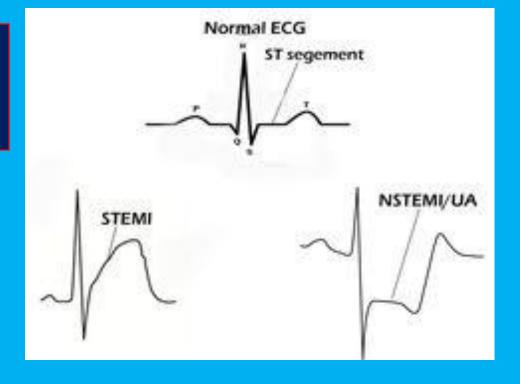




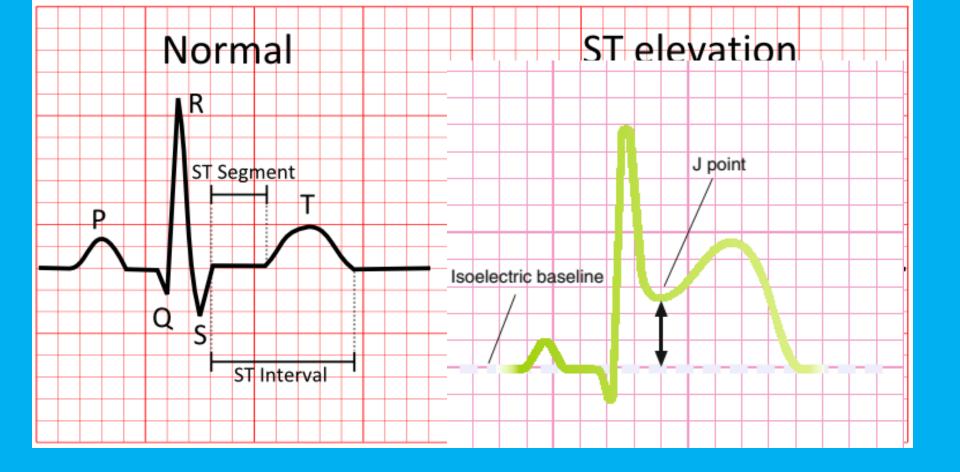




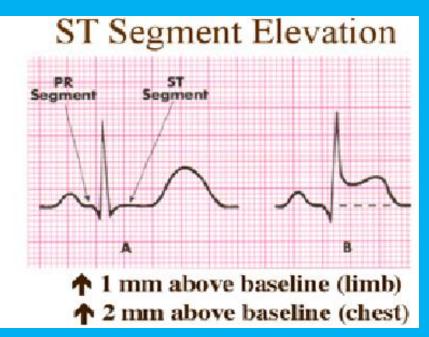
ST segment may be in Iso-electric line Elevated Depressed

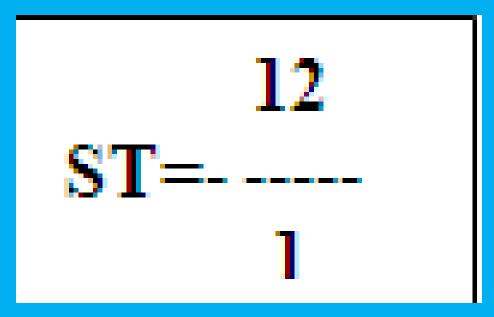


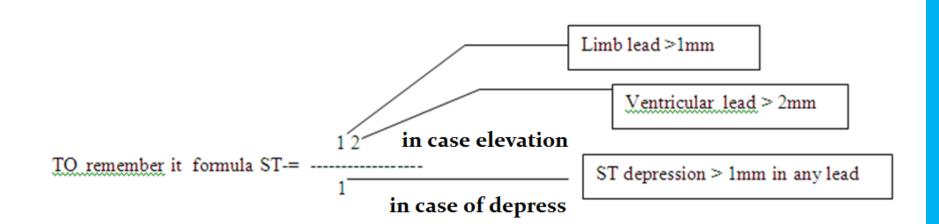




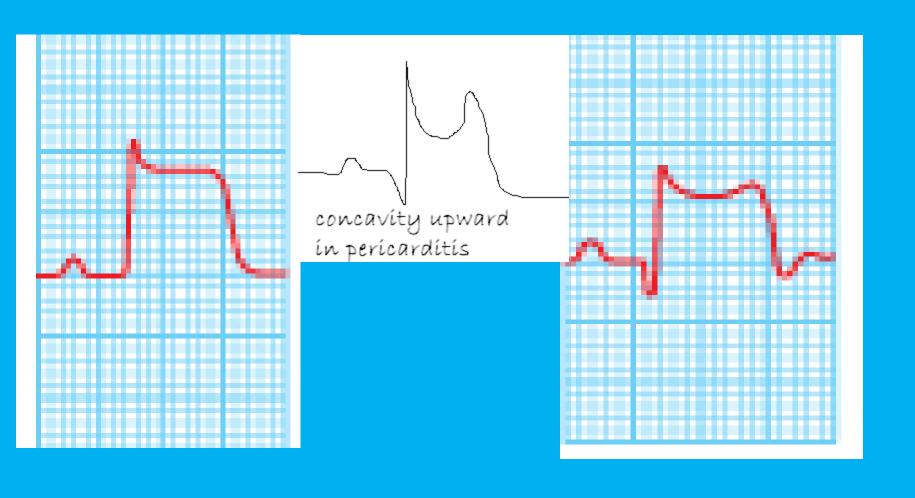
- •ST segment measure from end S to beginning of T wave
- •Normally it lies in Iso-electric line

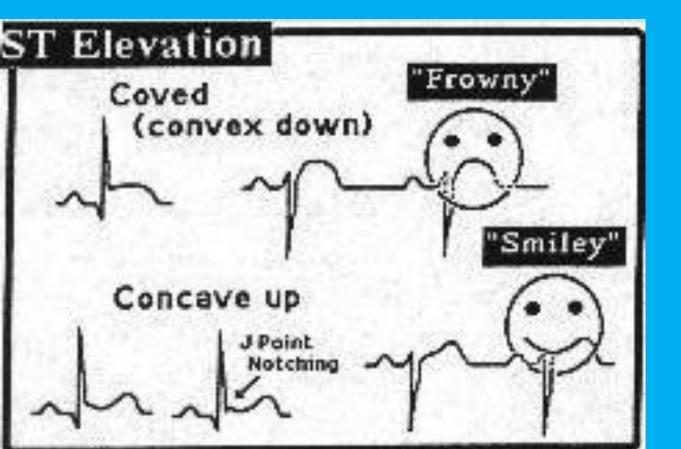






ST elevation should be consider if its height is More than 1 mm in limb lead More than 2 mm in ventricular lead





DD of ST elevation

- AMI (convexity up ward)
- Acute pericarditis (concavity up ward)
- Prinzmetal angina
- Ventricular aneurism (persistent ST elevation)
- Early repolarization (high take off)

How will u differentiated from ST elevation from early repolarization?

In MI

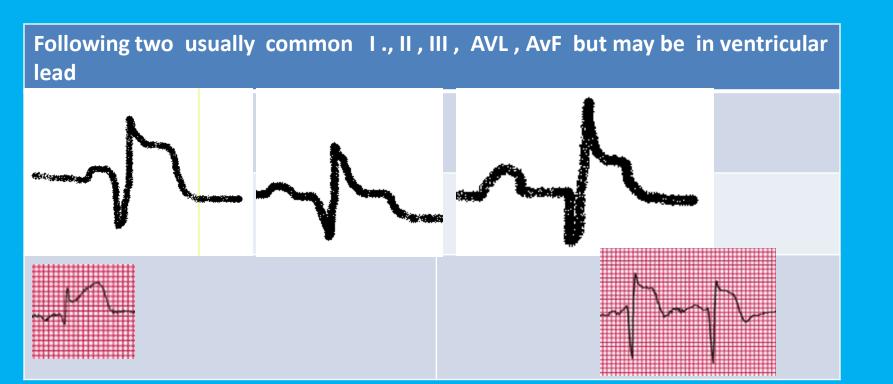
There is T inversion and Q wave

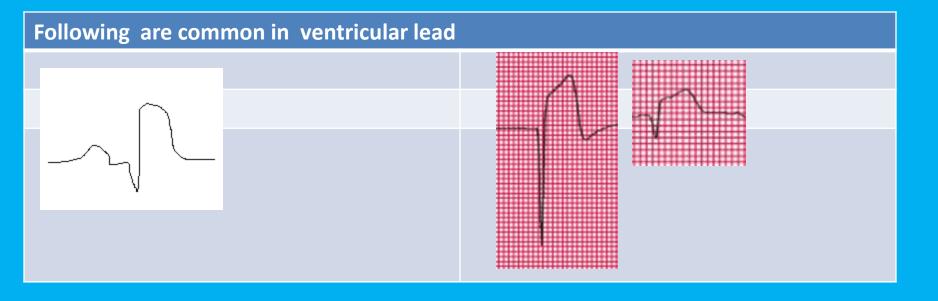
In early reploraization (ER)

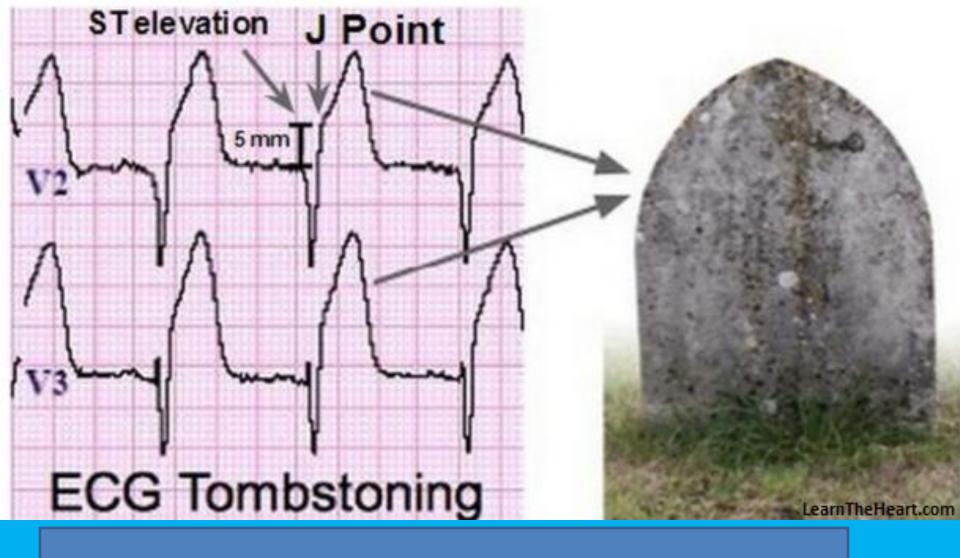
- •In young pt
- •Usually in V₄₋₆, with J point elevation
- No Q or T change

If u r still confused then do serial ECG that will show

- No change in ER
- •But in Case of MI, Q or T change will appear





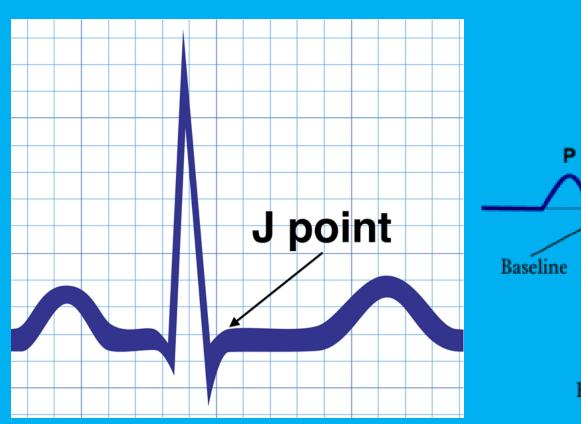


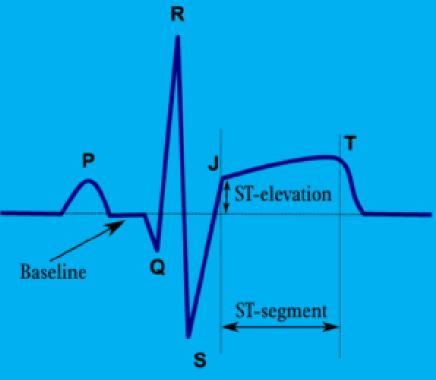
Typical pattern of ST elevation in anterior MI

J-POINT &

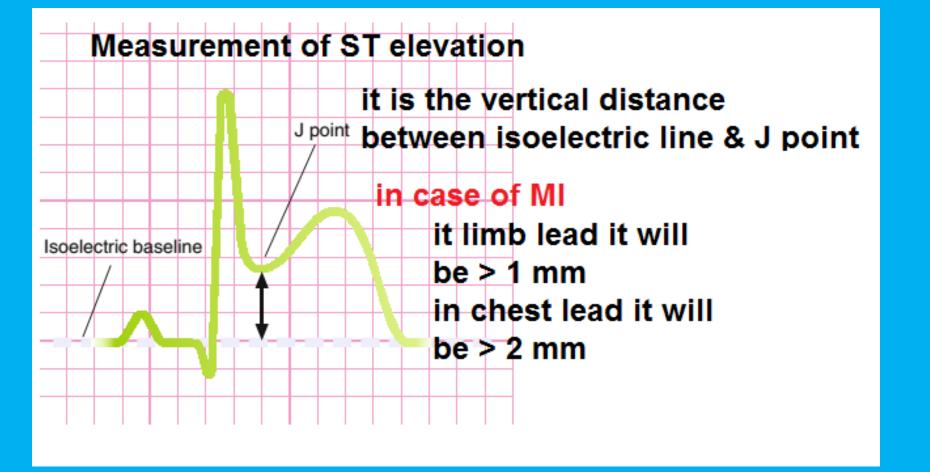
ST—ELEVATION DUE TO EARLY REPOLARIZATION

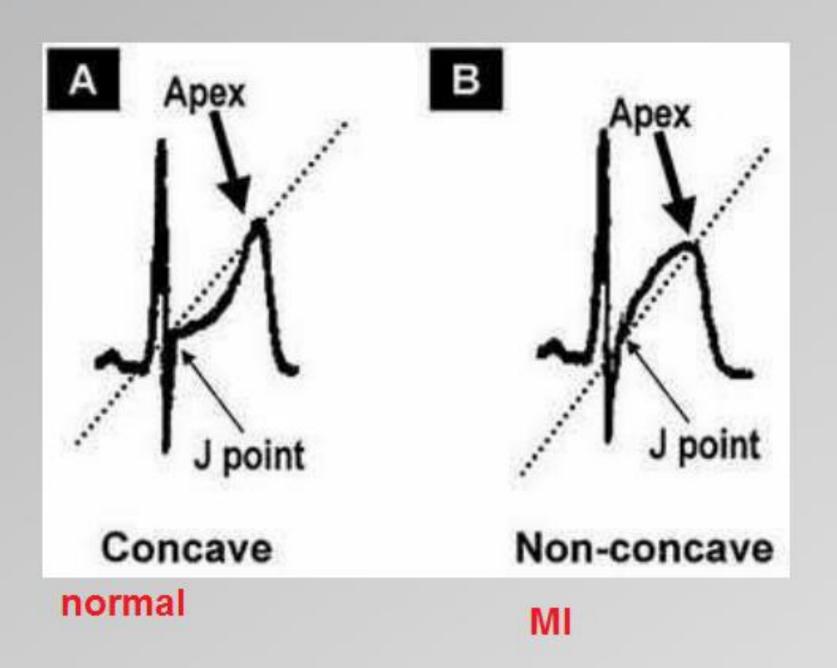
J point. The J point is the the *junction* between the termination of the QRS complex and the beginning of the ST segment



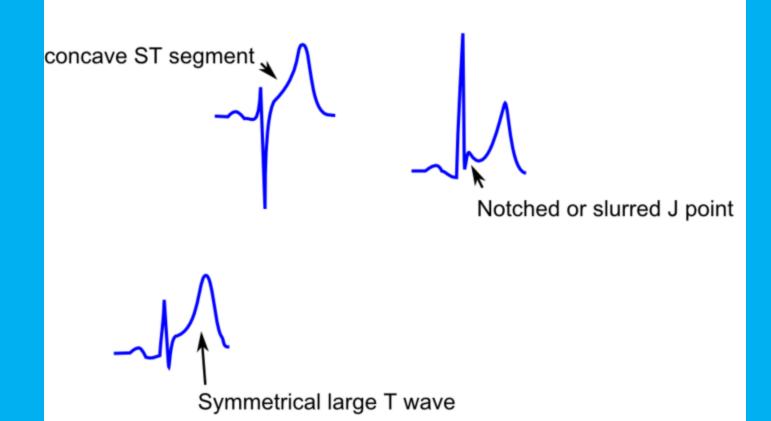


How to measure ST elevation?

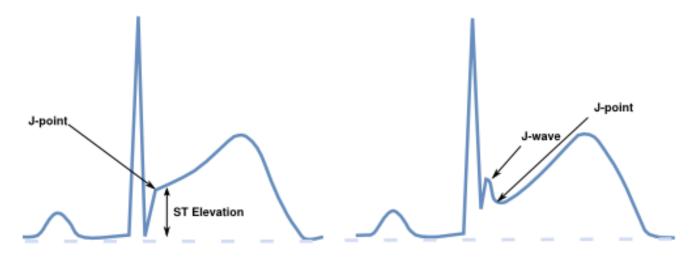




ST elevation due to early repolarization

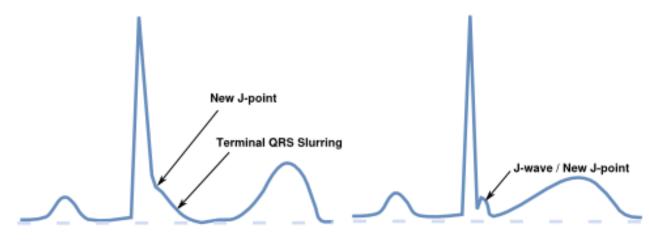


Classic Definition of Early Repolarization:ST Elevation

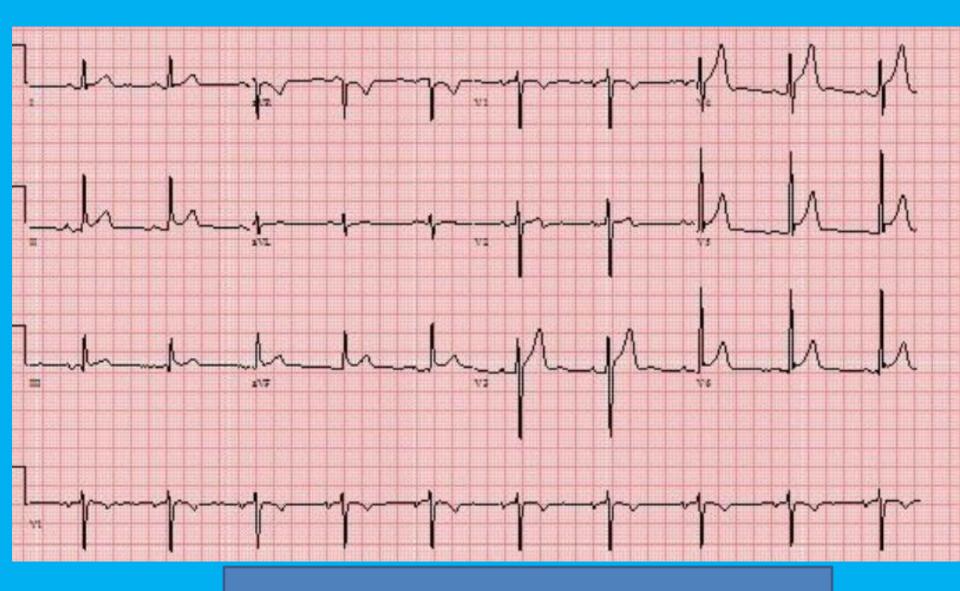


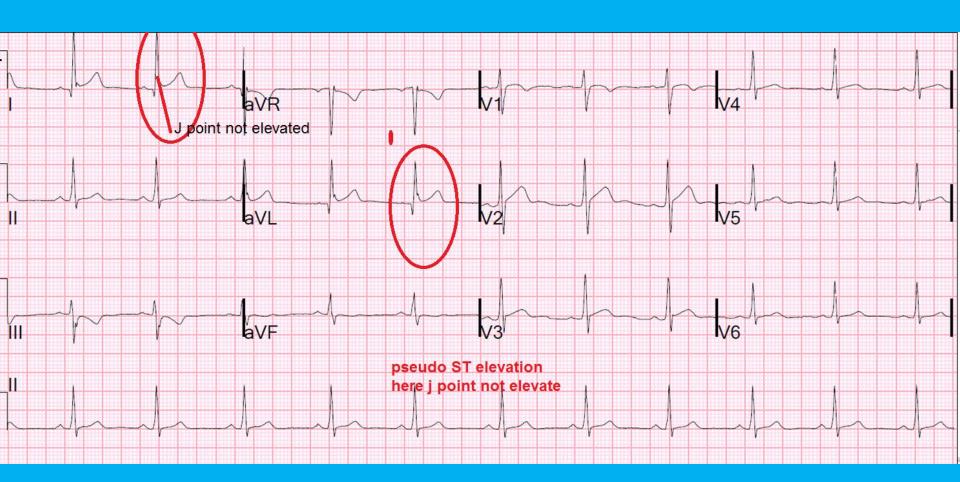
Classic Early Repolarization Without a J-wave Classic Early Repolarization With a J-wave

New Definitions of Early Repolarization



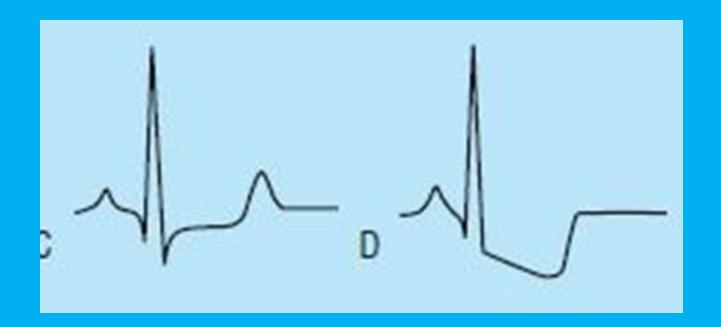
J-wave or the new "J-point Elevation" without STE



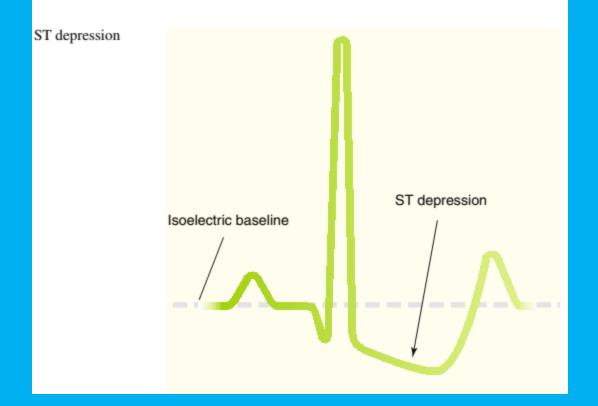


<u>Early repolarization</u> is a term used for ST segment elevation without underlying disease. It probably has nothing to do with actual early repolarization. It is commonly seen in young men. It is important to discern early repolarization from ST segment elevation from other causes such as <u>ischemia</u>. Characteristics of early repolarization are:

- ➤an upward concave elevation of the RS-T segment with distinct or "embryonic" J waves
- >slurred downstroke of R waves or distinct J points or both
- ➤RS-T segment elevation commonly encountered in the precordial leads and more distinct in these leads
- rapid QRS transition in the precordial leads with counterclockwise rotation
- persistence of these characteristics for many years
- ➤ absence of reciprocal ST depression
- ➤ large symmetrical T waves



ST---DEPRESSION

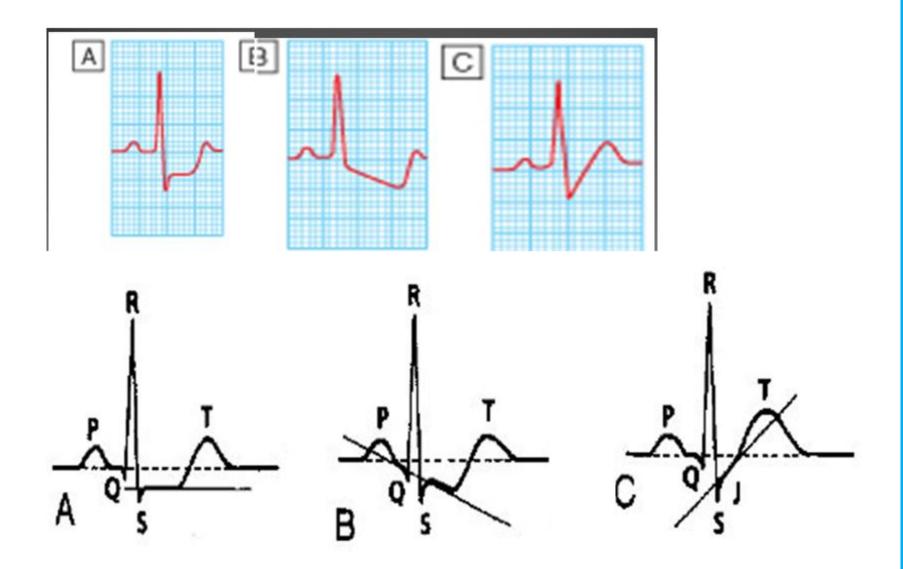


Cause of ST depression

- Myocardial ischemia (down / upward sloping)
- Ventricular hypertrophy with strain
- Digoxin toxicity (reverse tick mark)

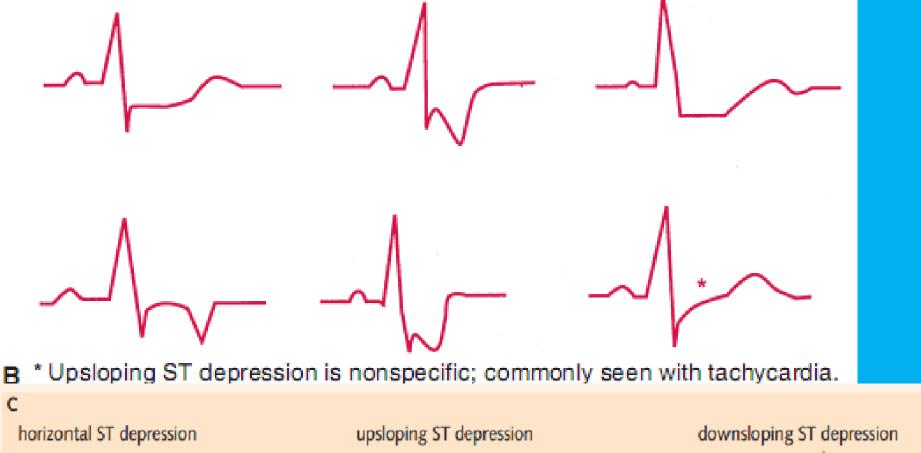
ST depression will significant if it found in two correspond lead such corresponding leads (I+AVL, II+ III+ AVF, V $_1$ to V4 $_{\rm ETC}$).

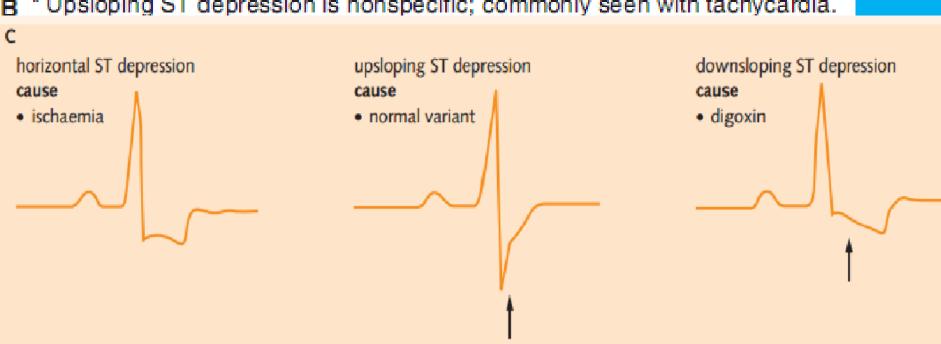
ST depression / T inversion present but not ischemia LVH / RVH
Bundle branch block (RBBB / LBBB)
Ectopic beat

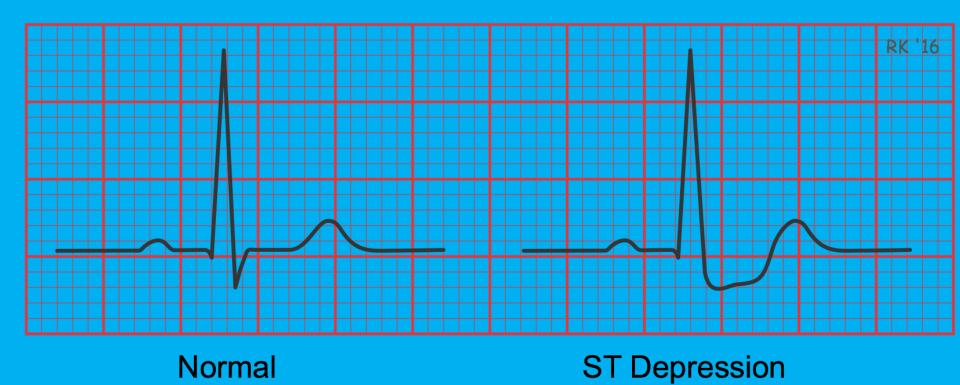


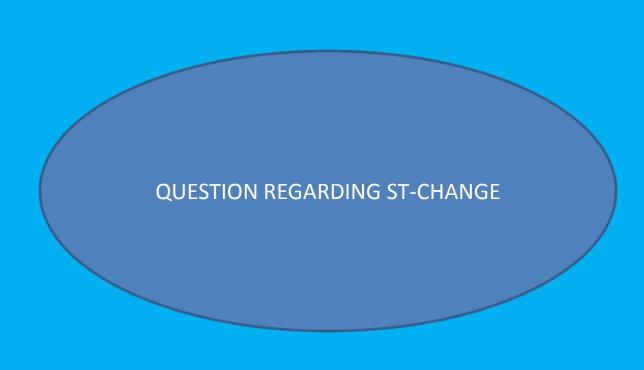
ST depression

- If ST segment is more 1 mm below from Iso-electric
- It indicate mainly myocardial ischemia









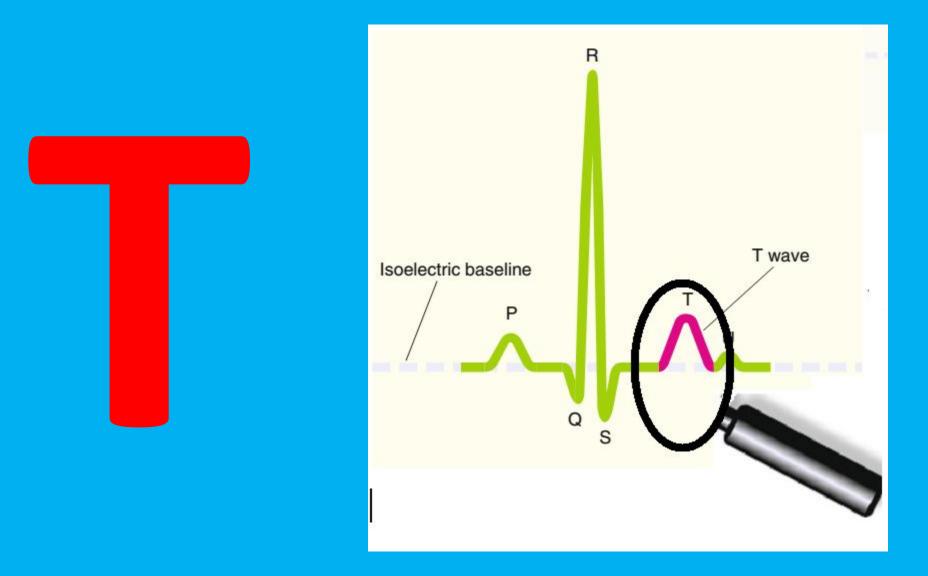
ST elevation:

- 1. Acute myocardial infarction
- 2. Acute pericarditis
- 3. Prinzmetal's angina (Non-infarction transmural ischemia)
- 4. Normal variant (Early repolarization pattern)
- 5. Ventricular aneurysn

ST depression:

- 1. Acute coronary syndrome
- 2. Angina pectoris
- 3. Ventricular hypertrophy with strain
- 4. Acute true posterior MI (in V1 and V2)
- 5. Digoxin toxicity





T-WAVE

In a ECG U got T wave in following way

T—inversion

T—tall T

T-flat

T- is normal if its height > 2mm and if its tip will be rounded and dome shape

$$T = 5 / 10$$

it means

T will be tall if its height > 5 mm in limb leads and

> 10 mm in ventricular lead

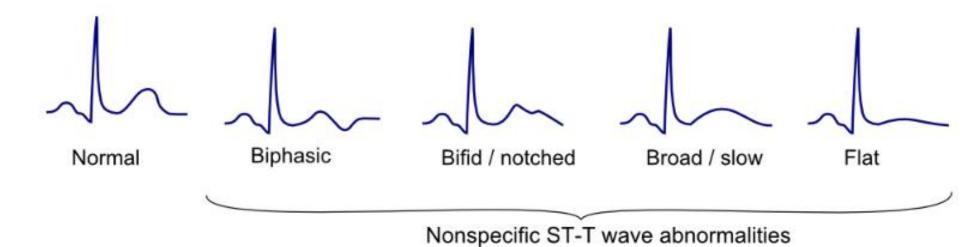
Remember this that tall and tent shape T found in hyper kalaemia

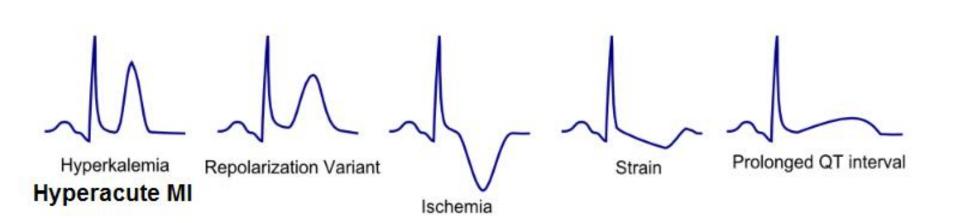
Cause small T or flat

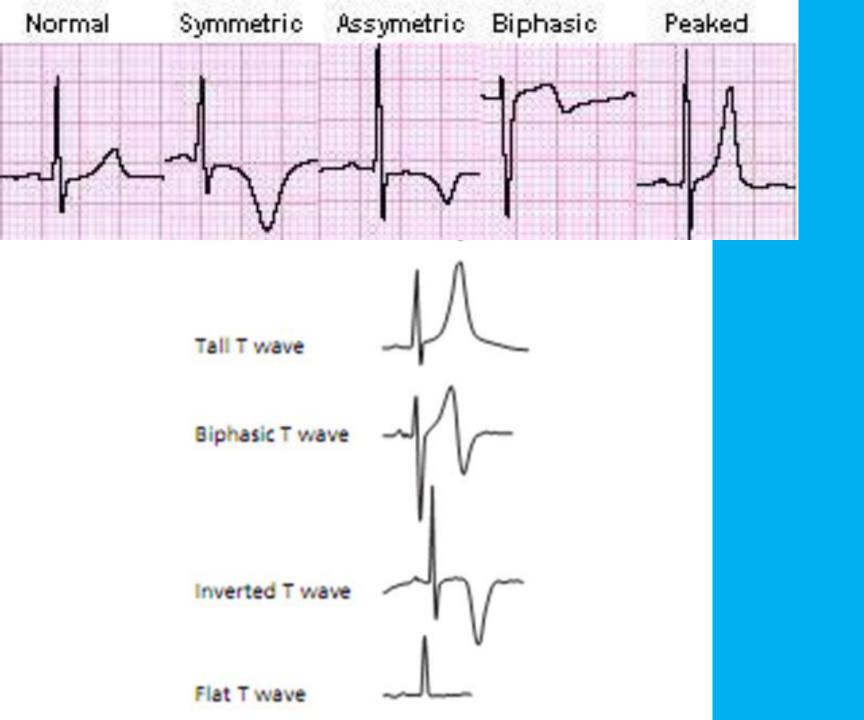
- In hypokalaemia
- Hypothyroidism
- Pericardial effusion

T –inversion will be significant if it found in two correspond lead such (I+AVL, II+ III+ AVF, V₁₊V₂).

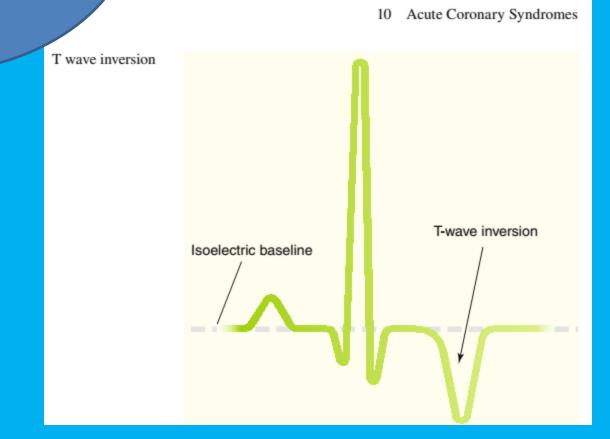
T wave morphology







Inverted "T"



Cause of T inversion

ST-MI

Non Q wave MI

Ischemia

Ventricular hypertrophy with strain

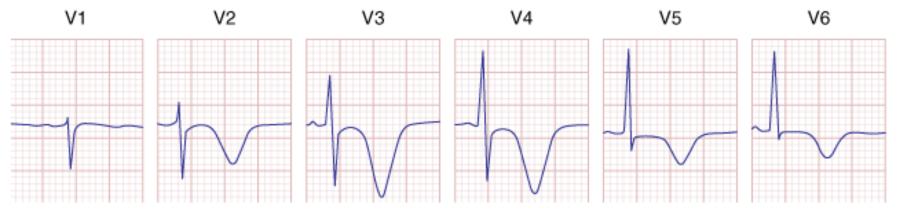
Ventricular ectopic

Bundle branch block

Cardiomyopathy

Digitalis

Symmetrical T in version if all lead $\underbrace{V_1 to}_6 V_6$. It is called sub endocardial MI



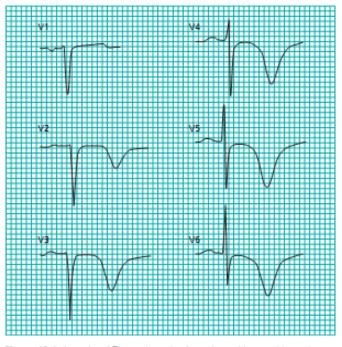
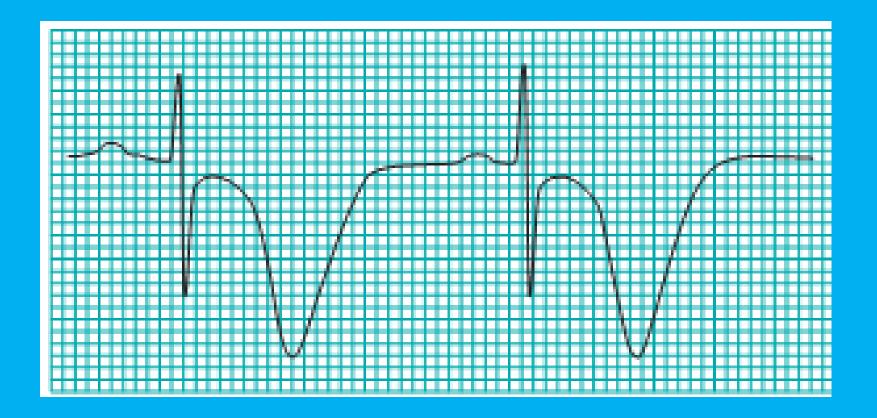
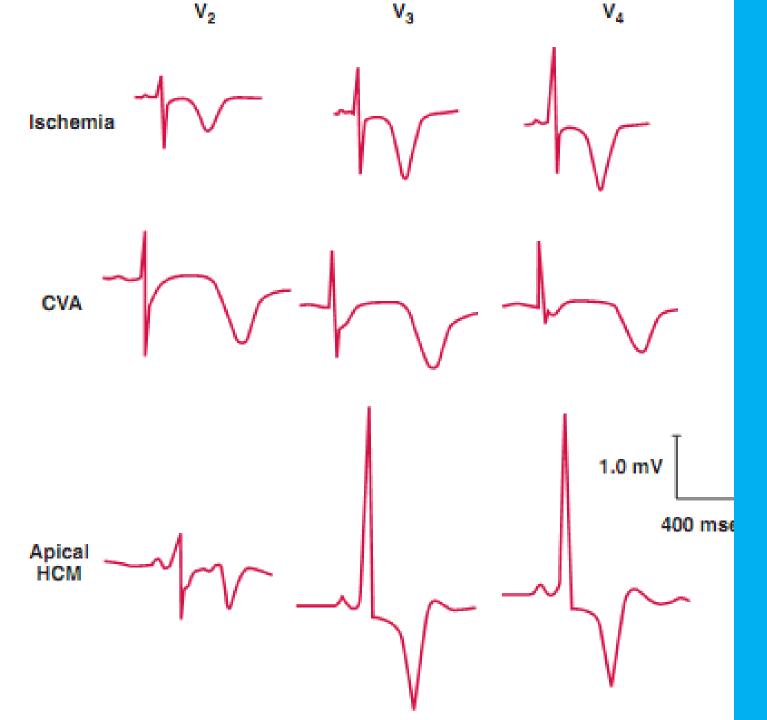


Figure 10.4 Arrowhead T wave inversion in patient with unstable angina



Massive T wave inversion and QT prolongation associated with subarachnoid haemorrhage.



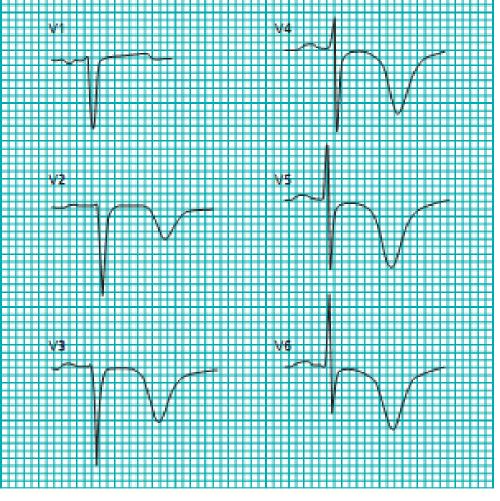


Figure 10.4 Arrowhead T wave inversion in patient with unstable angina.

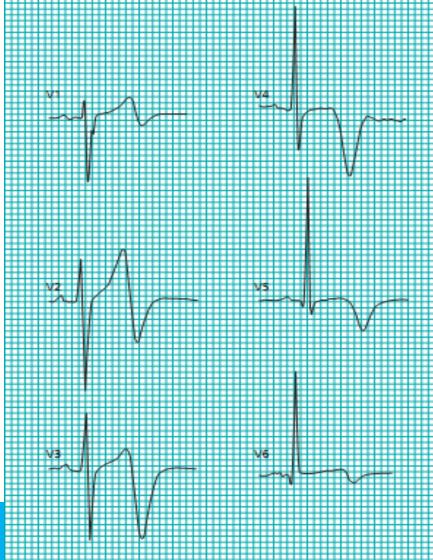
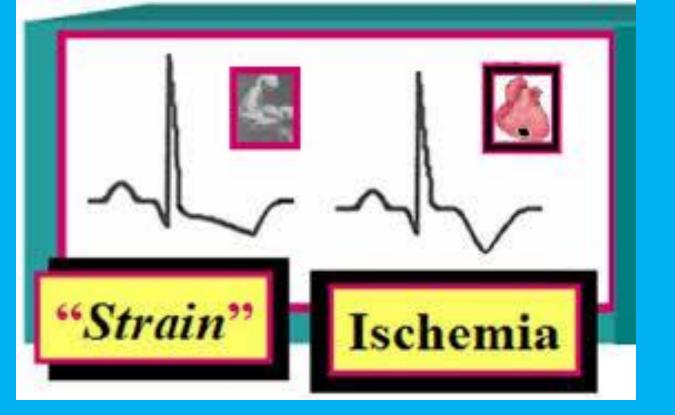
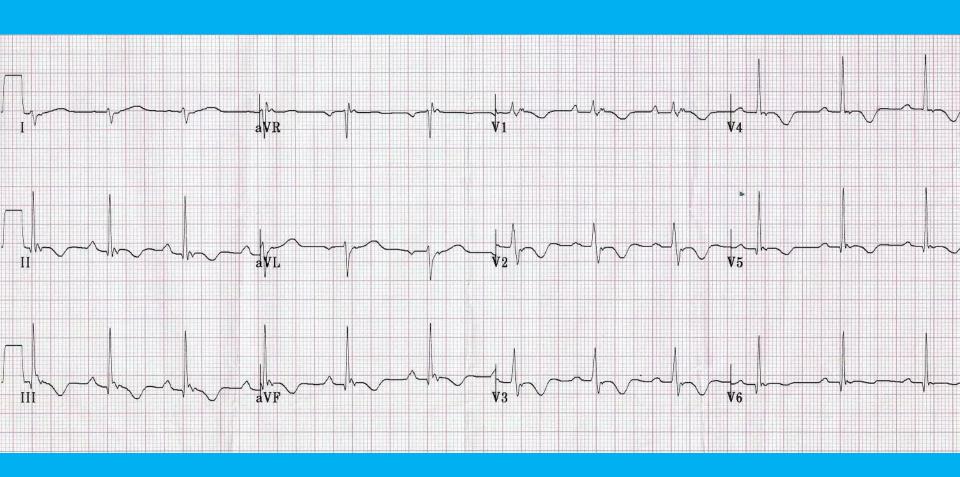


Figure 10.5 Biphasic T waves in man aged 26 with unstable angina.

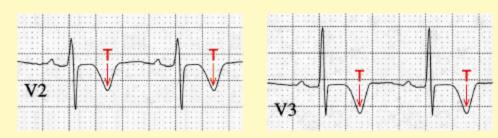




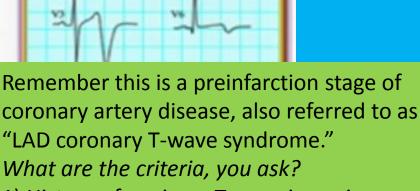
Wellens' Type 2

Wellens' Type 1

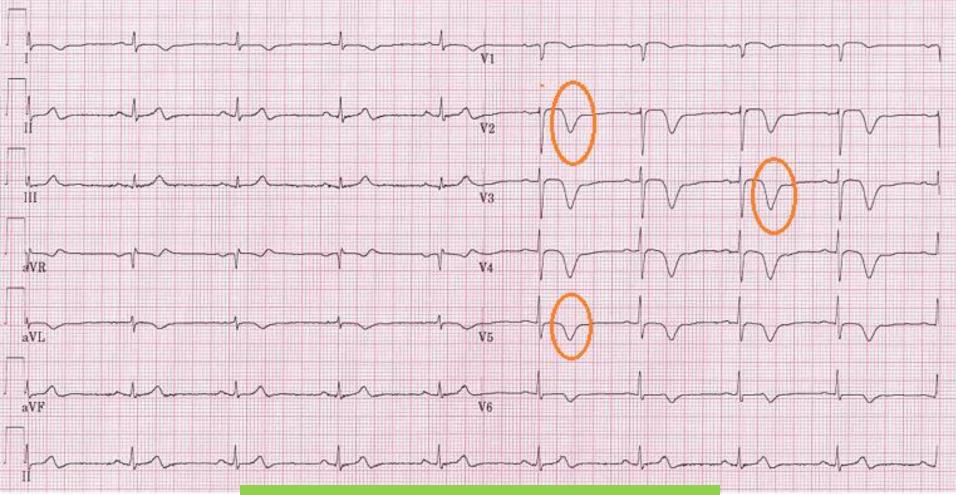
Wellens Syndrome



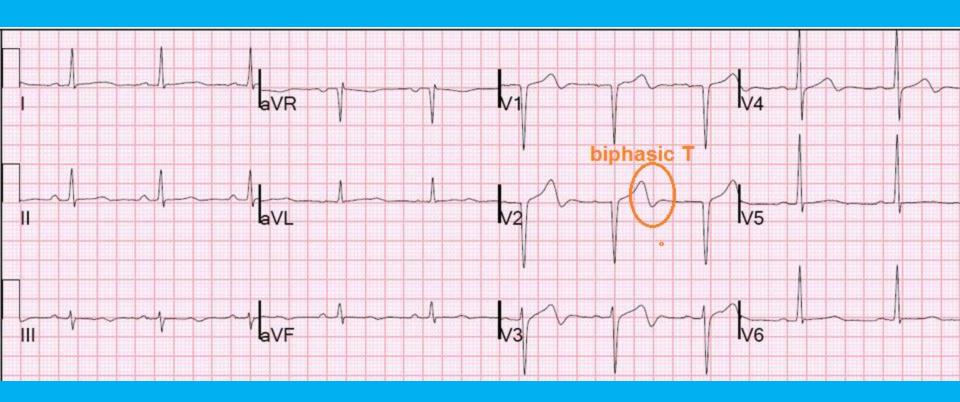
t Wellen's Type 1, there are deep T-wave inversions in V2 and V3. Now even if you didn't know this was called "Wellen's Sign," you would not send this patient home. They are coming in for admission, getting ASA, anticoagulation and cardiology consultation. These findings suggest to me a posterior wall AMI.

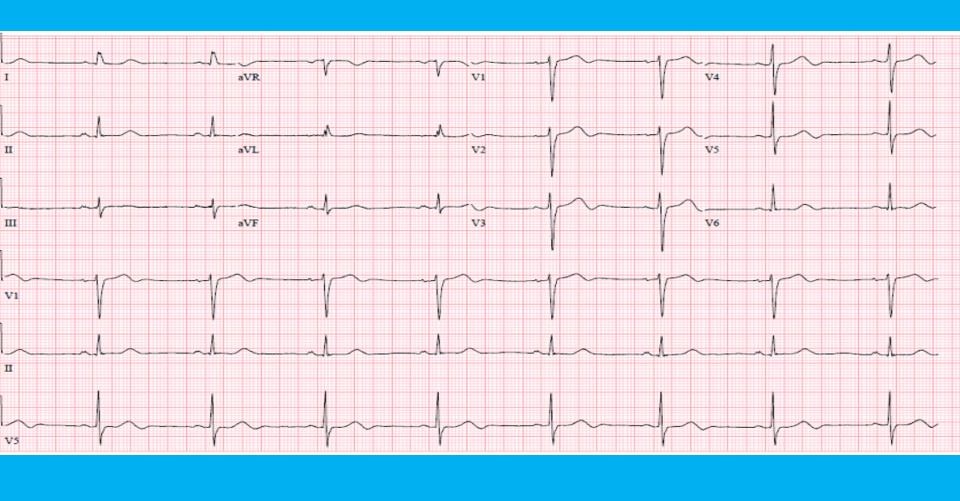


- 1) History of angina + T wave inversion or biphasic t waves in V2–V4
- 2) Normal or minimally elevated cardiac enzymes
- 3) No pathologic precordial q waves or loss of precordial R wave progression

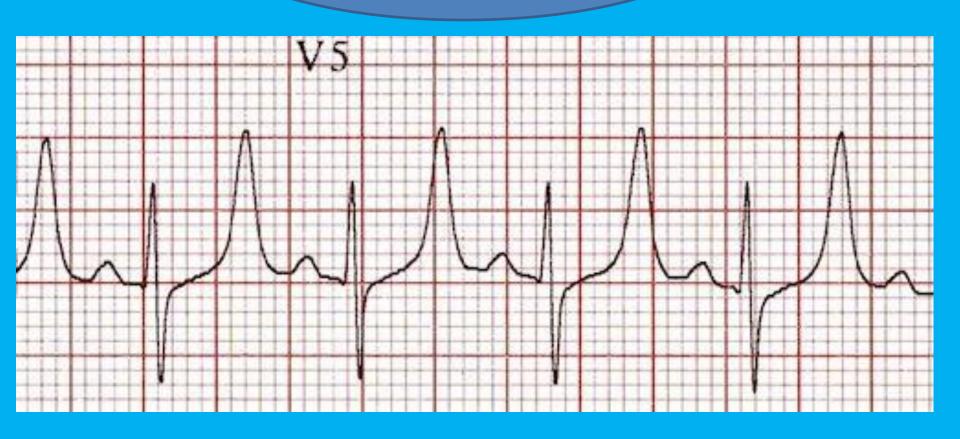


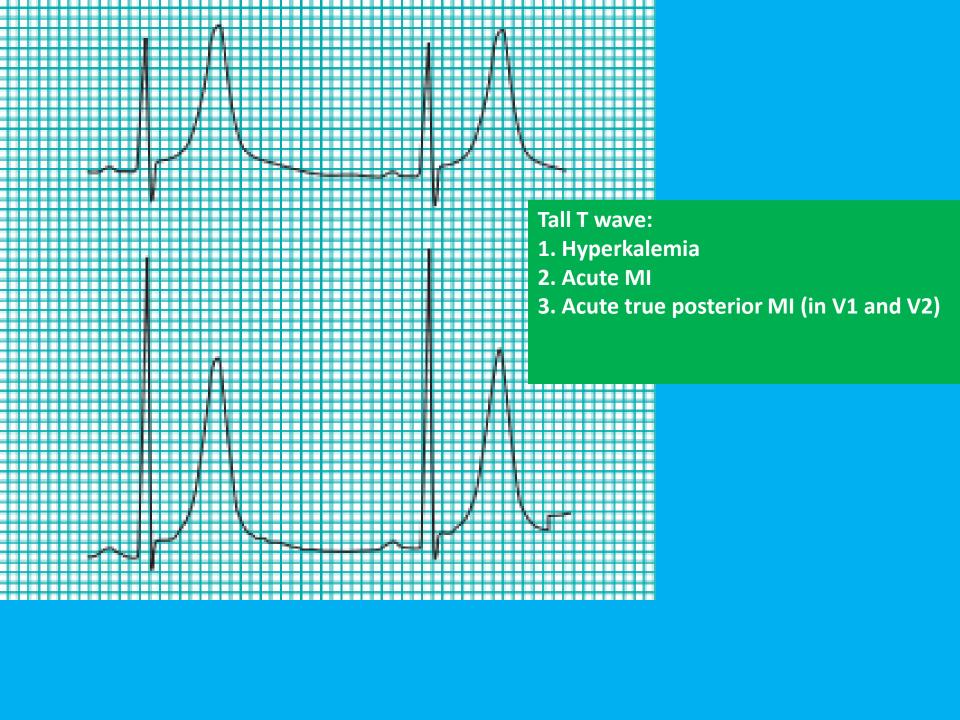
Wellens syndrome: symmetrical negative T wave in pre-cordial leads without R loss of R waves can regularly be observed in early anterior ischemia. Many patients with Wellens syndrome / sign turn out to have a critical proximal LAD stenosis







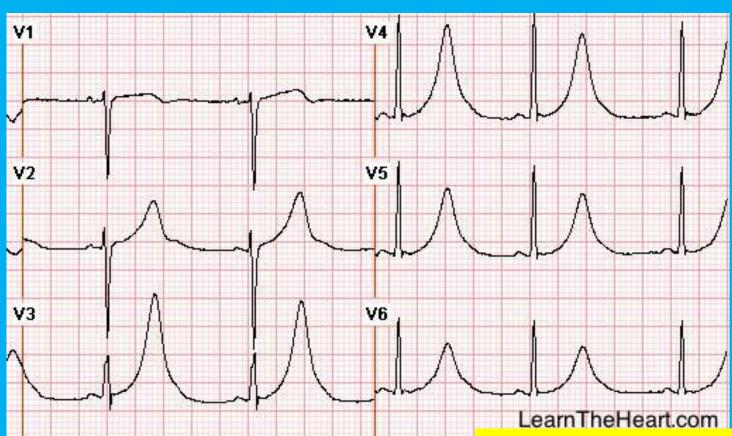




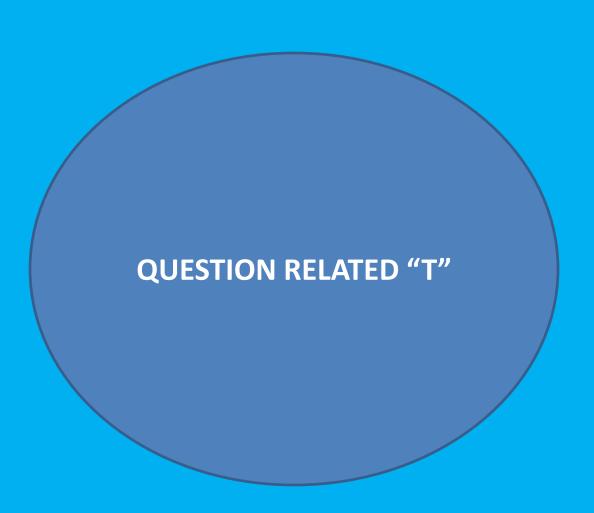


Tall T waves in leads V2 and V3 in patient with recent inferoposterior myocardial infarction, indicating posterior ischaemia.

Early change in MI



The first change ECG change during STEMI is "hyperacute T waves" which appear peaked and are related to localized hyperkalemia. These changes are rarely seen as they are transient and frequently occur prior to hospital arrival.



Tall T wave: 1. Hyperkalemia 2. Acute MI 3. Acute true posterior MI (in V1 and V2)	Small T wave: 1. Hypokalemia 2. Hypothyroidism 3. Pericardial effusion
T inversion: 1. MI 2. Myocardial ischemia 3. Subendocardial MI 4. Ventricular ectopic 5. Ventricular hypertrophy with strain 6. Acute pericarditis	

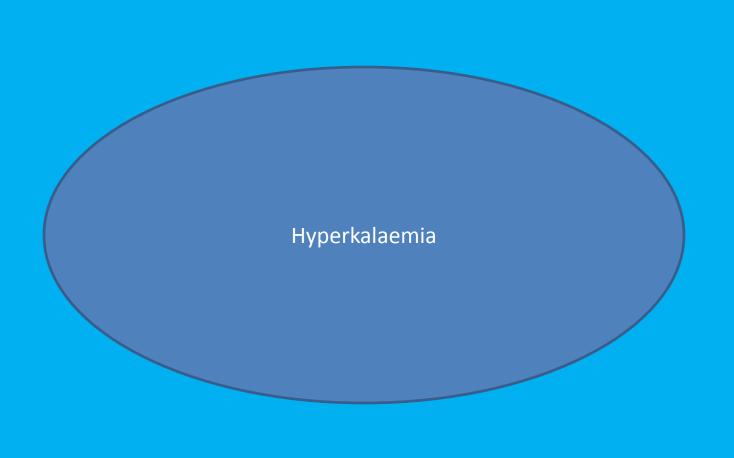
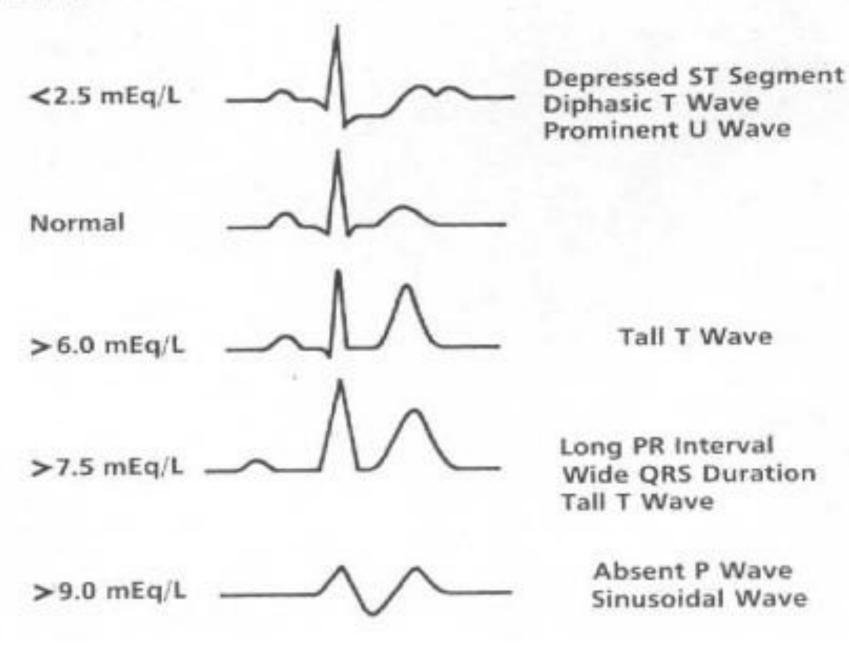




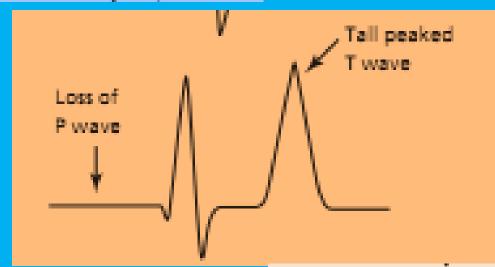
Table 9.1 The effects of hyperkalemia on the ECG dependent on level

Mild	Moderate	Severe
K+≥5.5 mmol/l	K+≥6.5 mmol/l	K+≥7.0 mmol/l
Tall peaked T waves Flat (low amplitude) P waves	Prolonged PR interval Wide P waves	Broad QRS complexes Ventricular arrhythmias
	No P waves	Asystole ST segment elevation

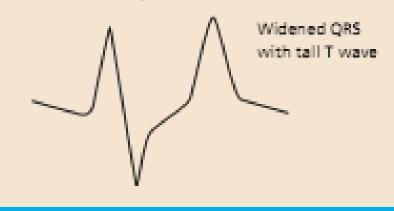
SERUM K







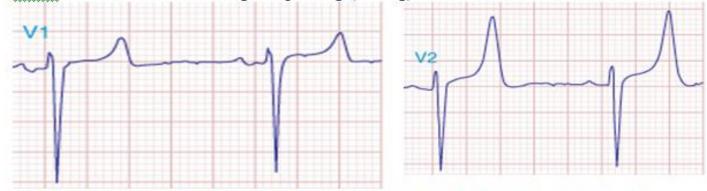
Serum potassium (mmol/l) Major change 5.5-6.5 -----Tall peaked T waves 6.5-7.5----- Loss of P waves 7.0-8.0 ------Widening of QRS complexes 8.0-10-----Sine wave, ventricular arrhythmias, asystole



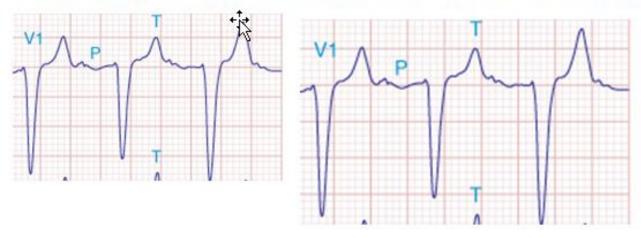


T: tall and tent shape T (>5 mm in limb lead /> 10 mm ventricular lead)

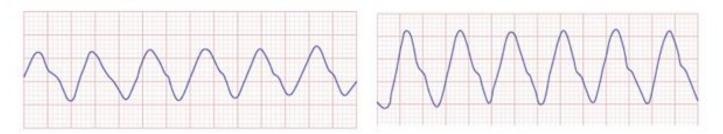
Mild: T become tall ,narrowing and peaking (tenting) of the T waves.

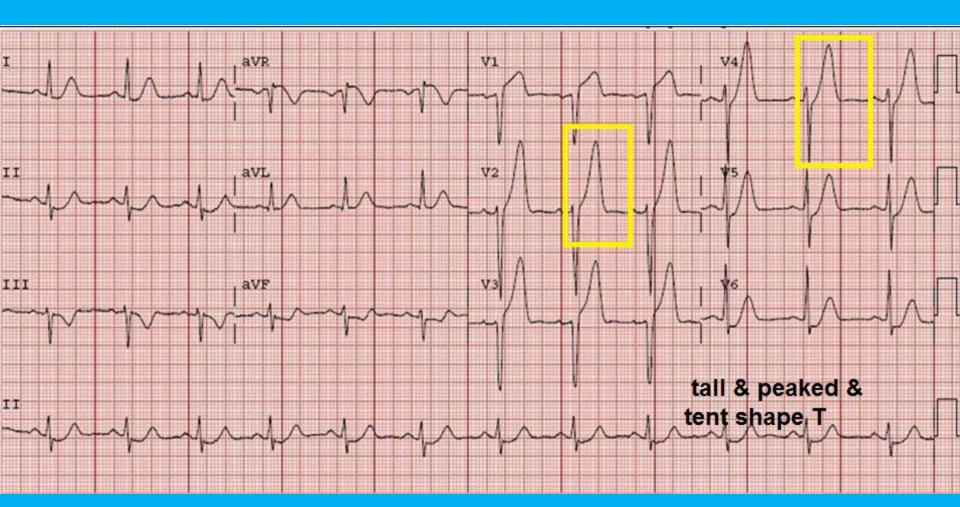


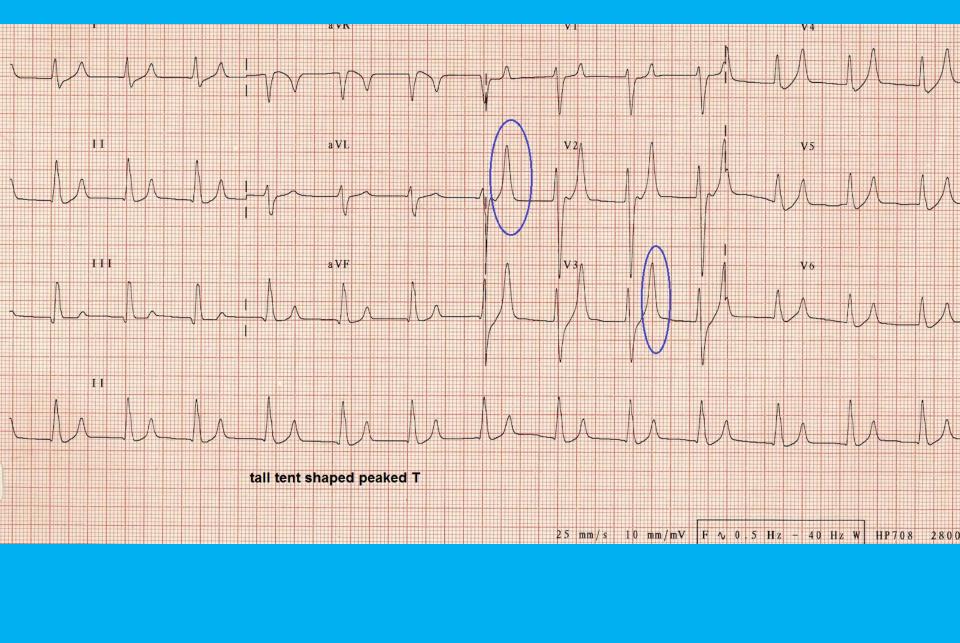
Further increase diminution in P-wave amplitude, and widening of the QRS interval.

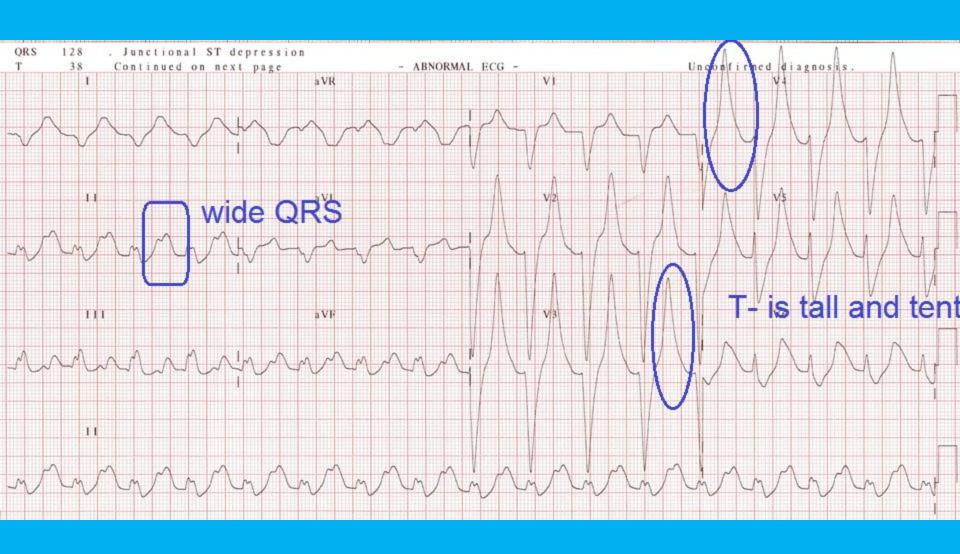


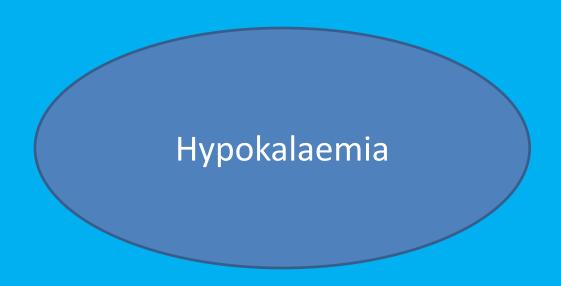
Severe hyperkalemia eventually causes cardiac arrest with a slow sinusoidal type of mechanism ("sine-wave" pattern) followed by asystole.

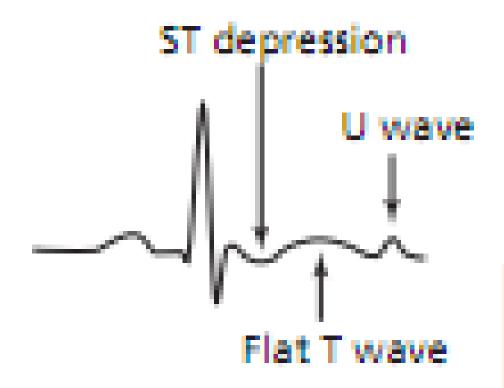


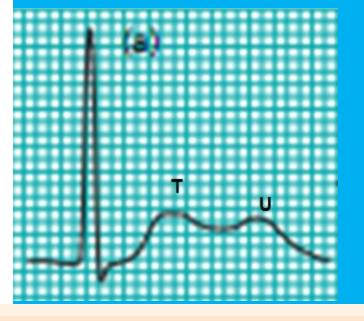


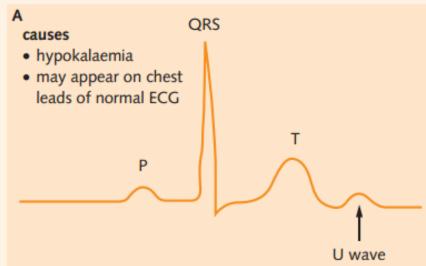








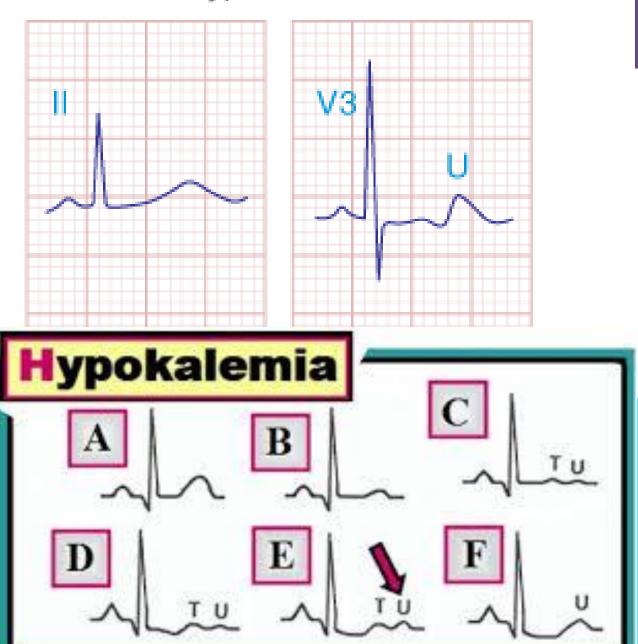




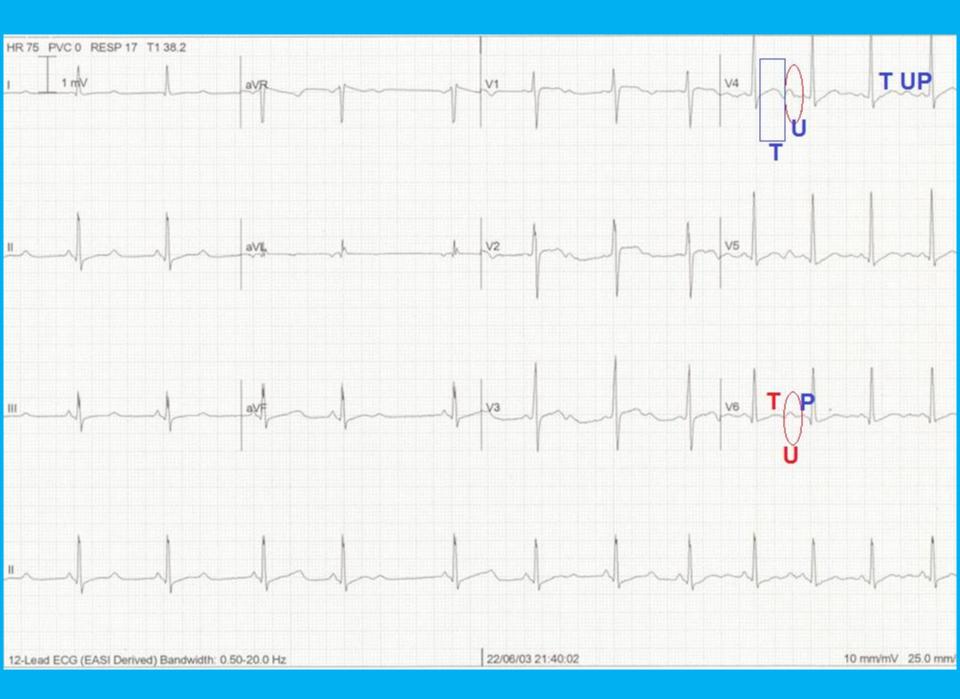
Electrocardiographic features of hypokalaemia.

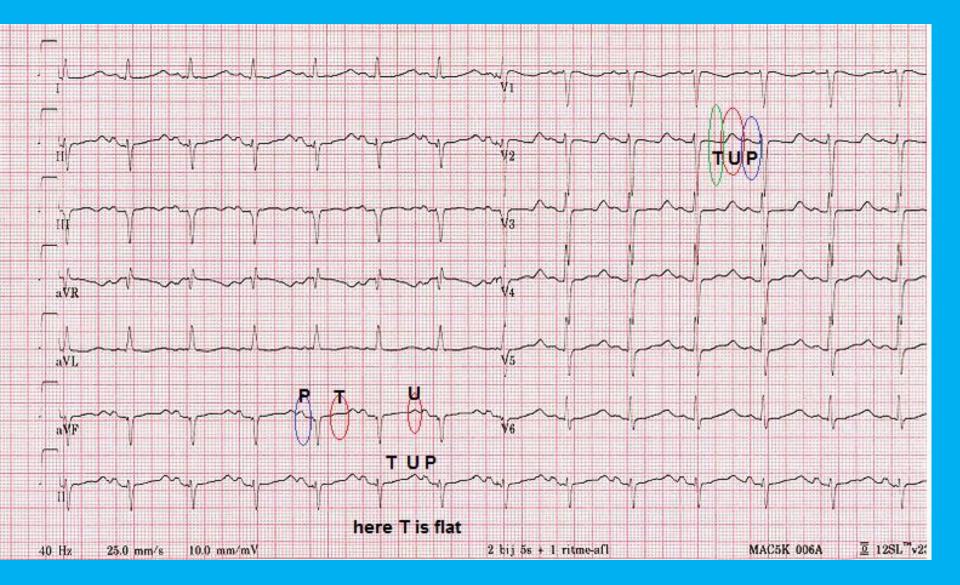
- •Broad, flat T waves
- •ST depression
- •QT interval prolongation
- •Ventricular arrhythmias (premature ventricular contractions, torsades de pointes, ventricular tachycardia, ventricular fibrillation)

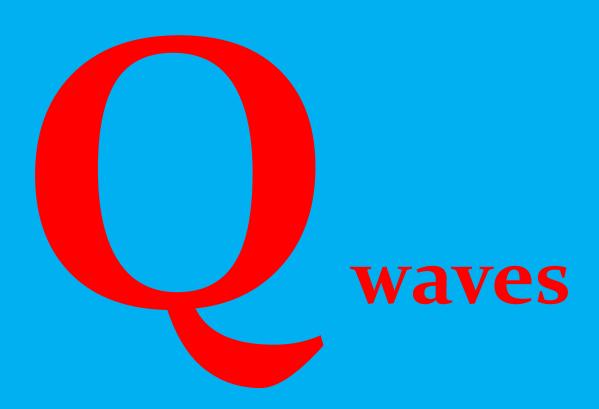
Hypokalemia

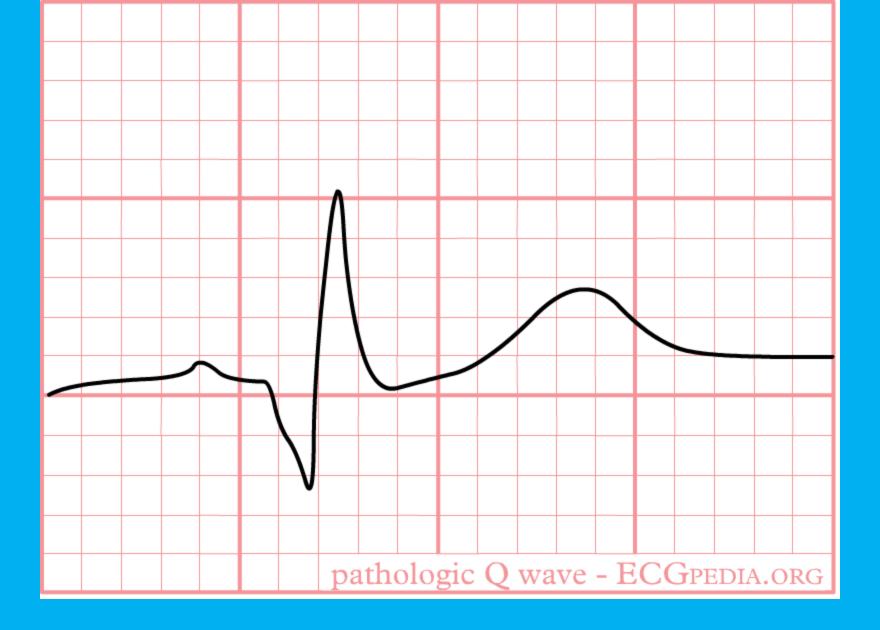


Hypo kalaemia: T become flat and appearance of U wave. Hypokalemia prolongs ventricular repolarization









Anatomy of Q wave: to remember DW = 21

Depth = 2 mm Wide = 1 mm

Criteria for pathological Q wave

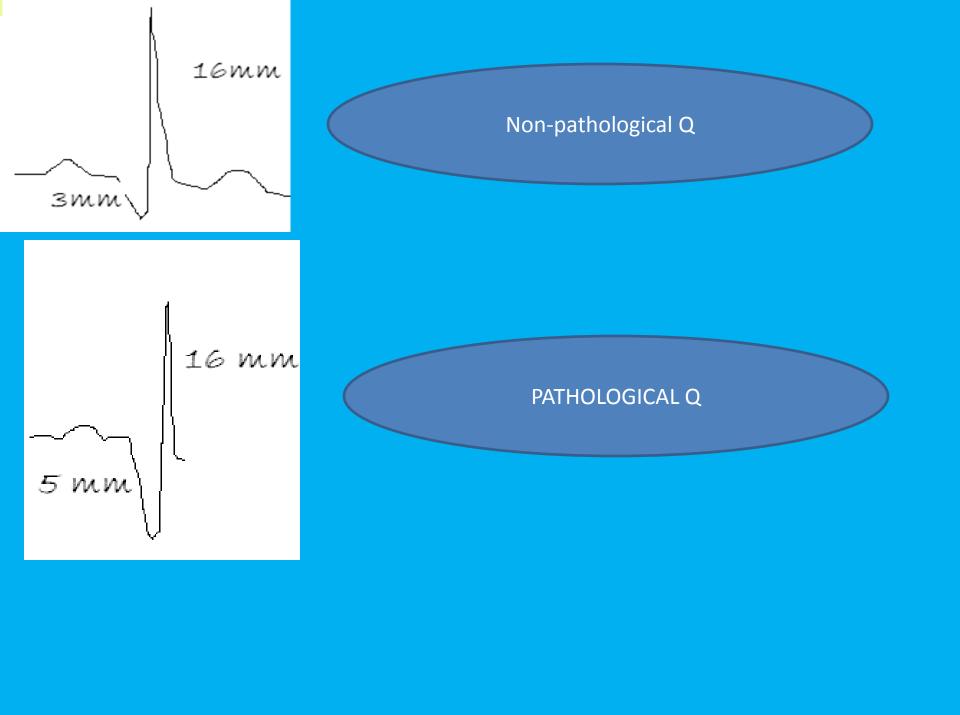
- Criteria 21 that means Depth > 2 mm and wide > 1 mm
- More than one corresponding leads (I+AVL, II+III+AVF, V₁₊V₂).
- 3. Q wave should be more than > 25 % of corresponding R wave height.
- Progressive loss of height of R wave.

Cause of Q wave in ECG

MI

- •LVH /RVH
- •LBBB
- Cardiomyopathy
- Pulmonary embolism

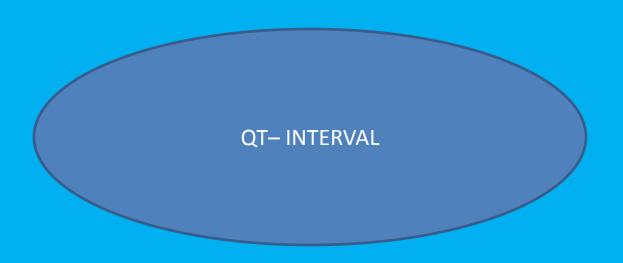
$$(Q_{III}S_{I}T_{III})$$



Q WAVE

Pathological Q wave:

- 1. MI
- 2. Left ventricular hypertrophy (in V1, V2 and V3)
- 3. LBBB
- 4. Pulmonary embolism (only in lead III)
- 5. WPW syndrome (in lead III and aVF)



U WAVE

Prominent U wave:

- 1. Normally present
- 2. Hypokalemia
- 3. Bradycardia
- 4. Ventricular hypertrophy
- 5. Hypercalcemia
- 6. Hyperthyroidism

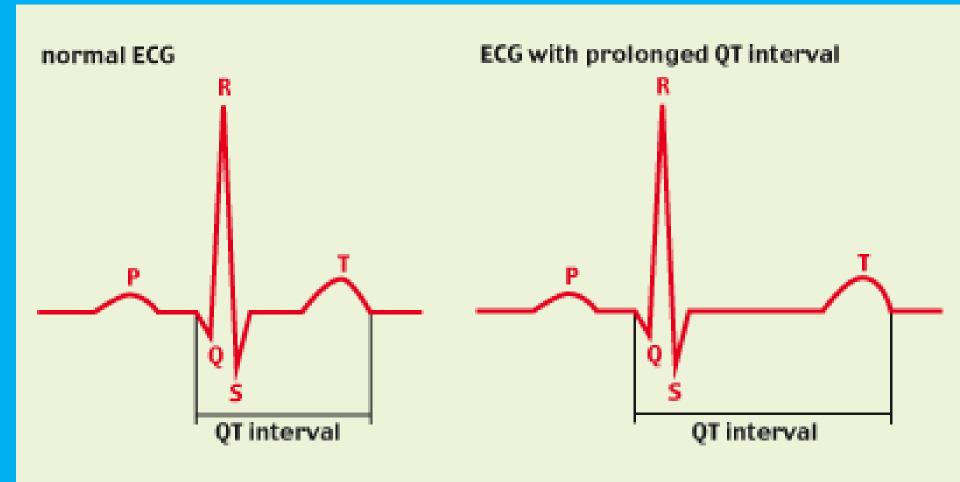
Short QT interval:

- 1. Tachycardia
- 2. Hyperthermia
- 3. Hypercalcemia
- 4. Digoxin effect
- 5. Vagal stimulation

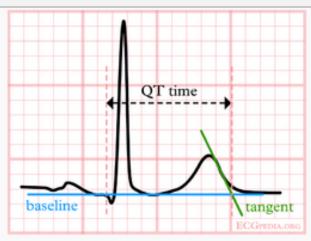
Long QT interval:

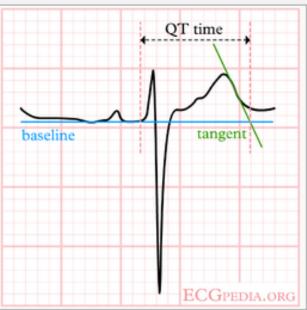
- 1. Bradycardia
- 2. Hypocalcemia
- 3. Acute MI
- 4. Acute myocarditis
- 5. Cerebrovascular accident
- 6. Hypertrophic cardiomyopathy
- 7. Hypothermia

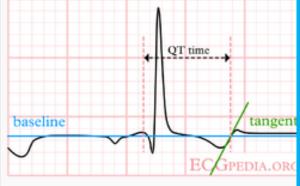
the **QT interval** is a measure of the time between the start of the Q wave and the end of the T wave in the heart's electrical cycle. The **QT** intervalrepresents electrical depolarization and repolarization of the ventricles.



How to measure QT if the QT segement is abnormal



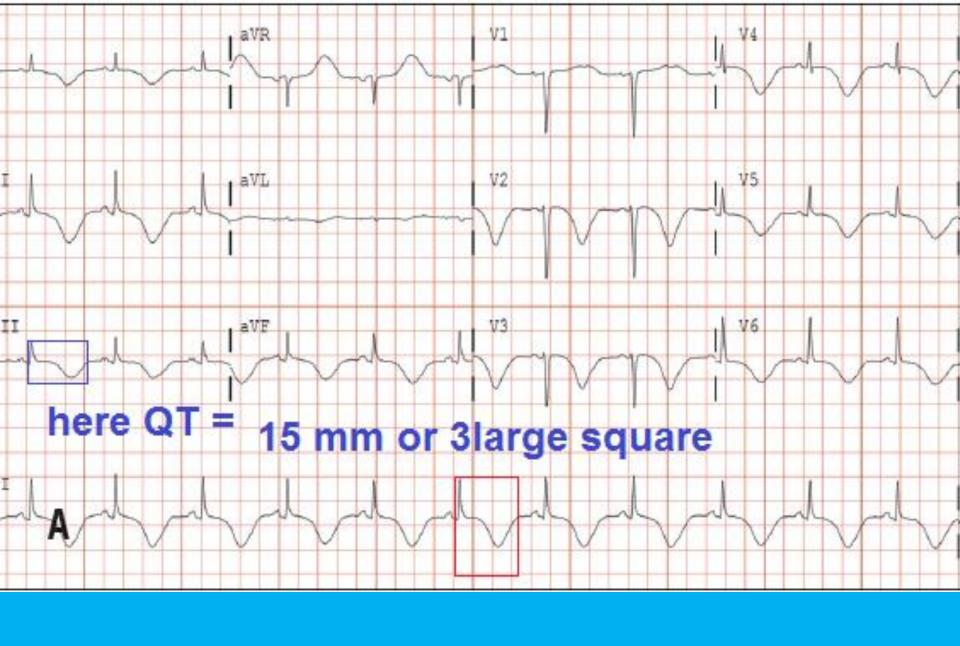




The T wave is broad, but the tangent crosses the baseline before the T wave joins the baseline. The QT interval would be overestimated when this last definition of the end of the T wave would be used

The ECG does not meet the baseline after the end of the T wave. Still, the crossing of the tangent and baseline should be used for measurements.

A bifasic T wave. The tangent to the 'hump' with the largest amplitude is chosen. This can change from beat to beat, making it more important to average several measurements.





Prolonged QTc Causes

Drugs

- Too many to actually list here, but some commonly used Rx in Peds are:
 - Antimicrobials
 - Macrolides (Erythromycin, Clarithromycin, Azithromycin)
 - Fluoroquinolones (Levofloxacin, Gatifloxacin, Ciprofloxacin)
 - Antifungals -(Fluconazole, Itraconazole, Voriconazole)
 - Bactrim (another reason not to use bactrim)
 - Antipsychotics (Amitriptyline, Desipramine, Imipramine, Sertraline)
 - Sedatives (Chloral hydrate)
 - ONDANSETRON (I know we have discussed it's value, but nothing is perfect)

Electrolyte Issues

- Hypokalemia
- Hypocalcemia
- Hypomagnesemia

Congenital Long QT Syndrome

- Rare, but real problem.
- Others
 - Hypothermia
 - Cardiac ischemia
 - Increased ICP

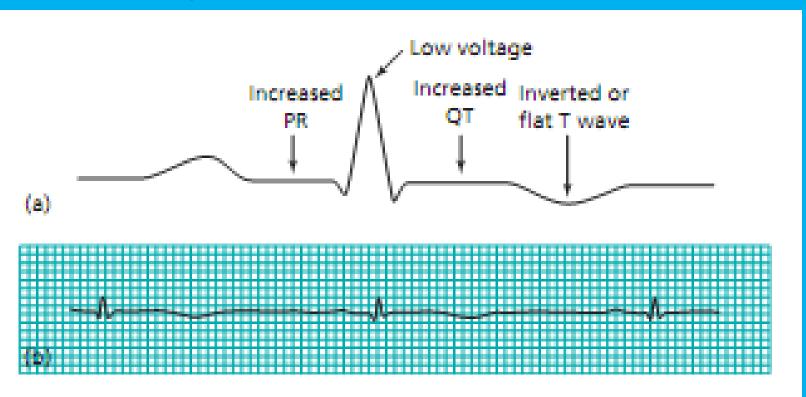
ECG FEATURE IN HYPOTHYROIDISM

Most common

- ❖Sinus bradycardia
- ❖ Prolonged QT interval
- ❖Flat or inverted T waves

Less common

- ➤ Heart block
- ► Low QRS voltages
- ➤ Intraventricular conduction defects
- ➤ Ventricular extrasystoles



ELECTROCARDIOGRAPHIC FEATURES OF THYROTOXICOSIS.

Most common findings

- oSinus tachycardia
- Olncreased QRS voltages
- OAtrial fi brillation

Other findings

- OSupraventricular arrhythmias (premature atrial beats, paroxysmal supra-
- oventricular tachycardia, multifocal atrial tachycardia, atrial fl utter)
- ONon-specific ST and T wave changes
- Ventricular extrasystoles

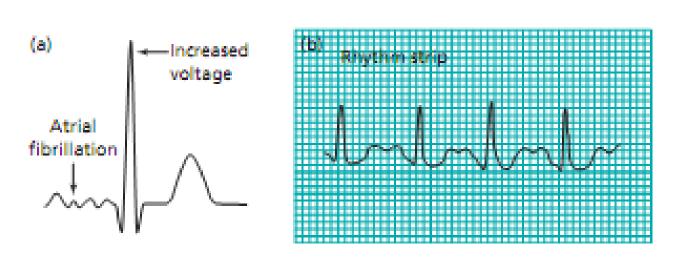
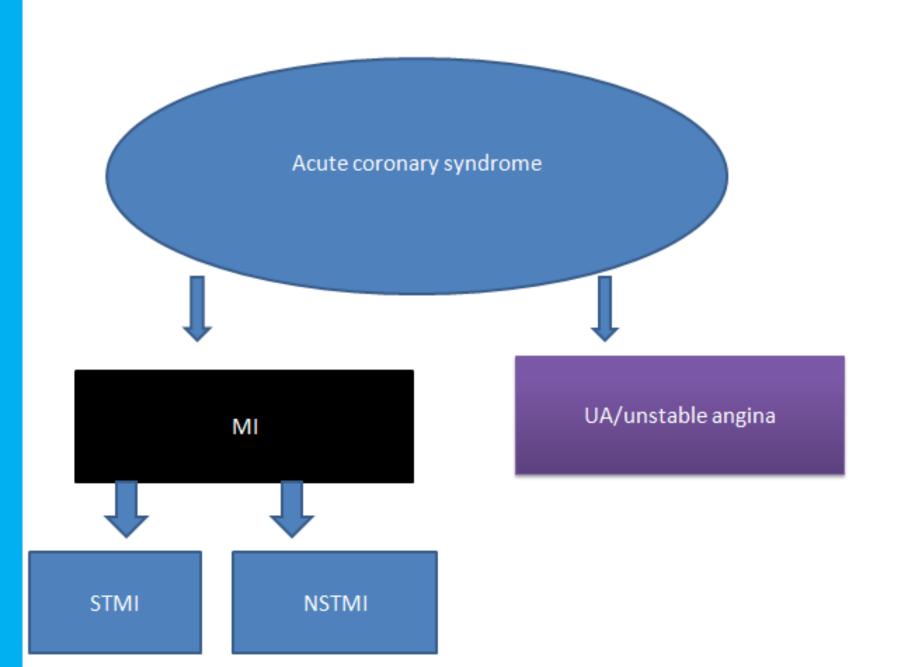
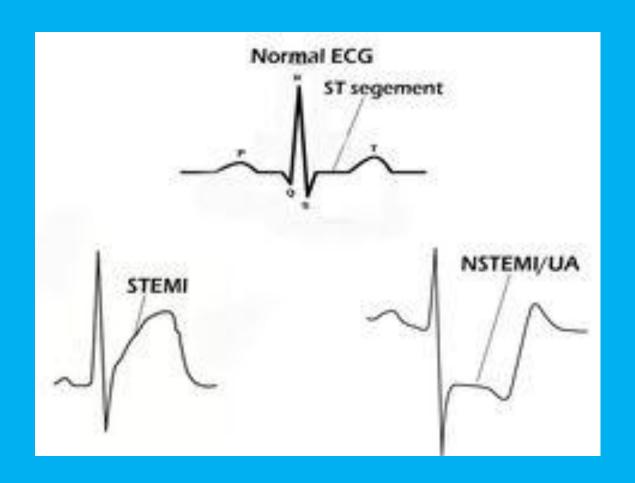


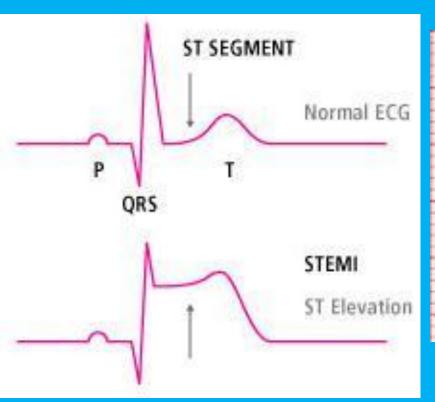
Figure 14.6 (a) Diagram of electrocardiographic changes associated with thyrotoxicosis. (b) Sinus tachycardia in patient with thyrotoxicosis.

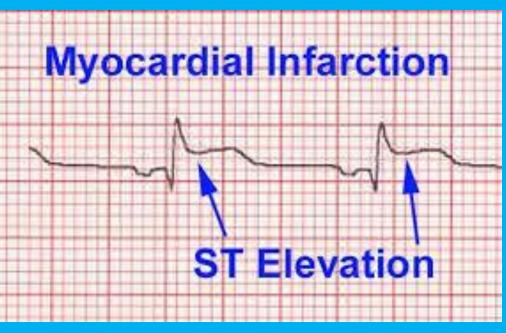
ACUTE CORONARY SYNDROME And Ischemia or ischaemic change in ECG

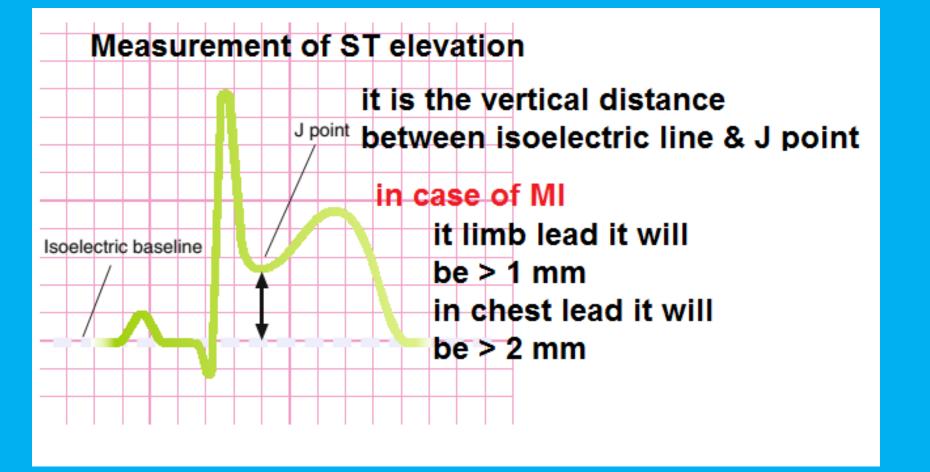




STMI



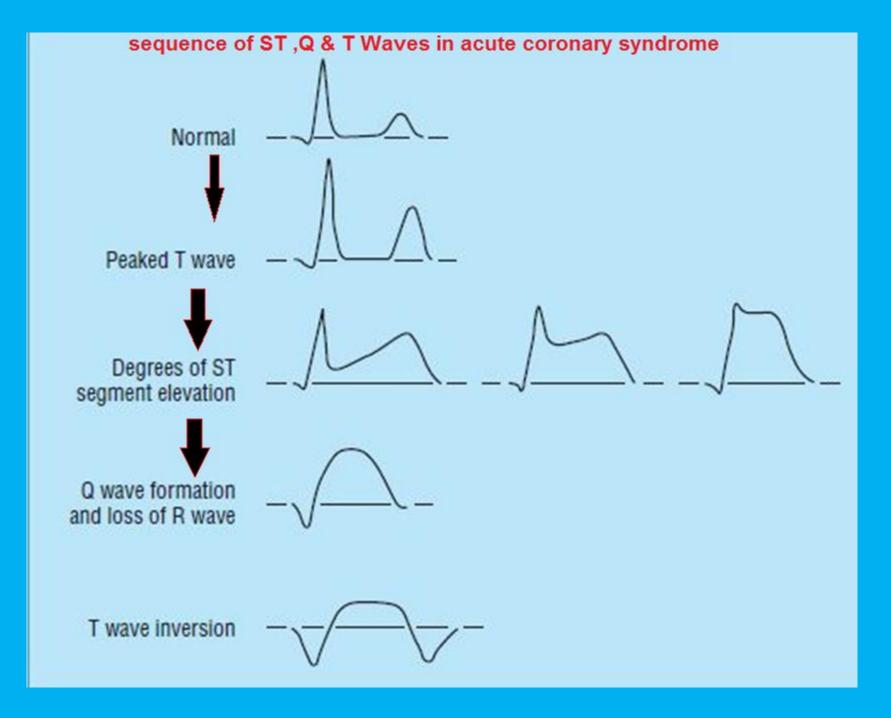




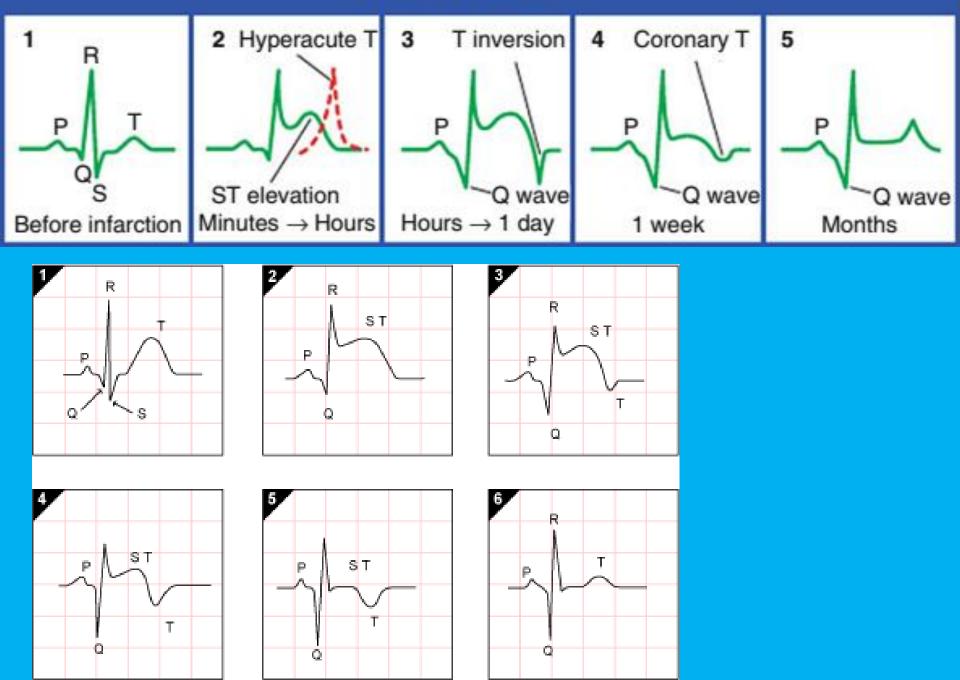
Causes of ST-Segment elevation

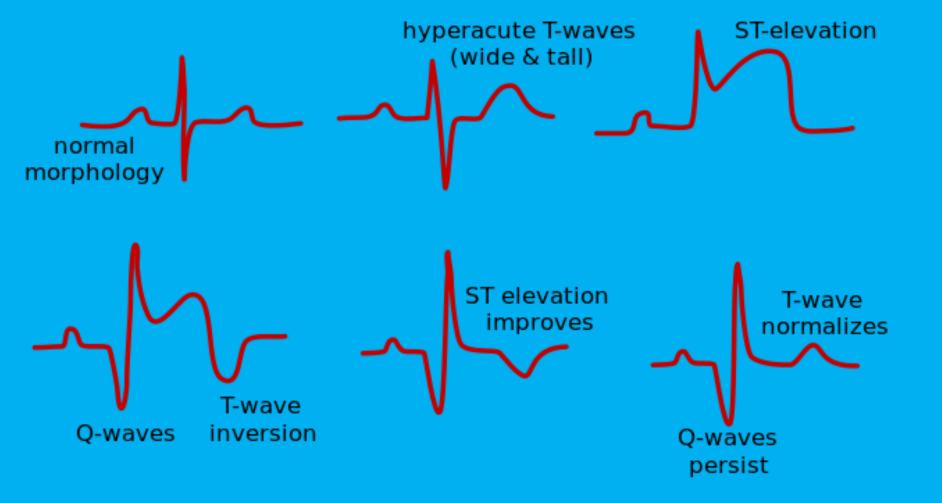
- Acute Myocardial Infarction (MI)
- Acute Pericarditis
- High take off
- Brugada syndrome
- Left ventricular aneurysm
- Prinzmetals angina
- Normal variant

Sequence of ST change, Q wave and T wave in acute coronary syndrome



Evolution of stemi





First Hyper acute T \rightarrow appearance of ST elevation \rightarrow then appearance of Q wave & T wave \rightarrow Decrease the height of R wave \rightarrow T wave become normalized \rightarrow Q wave persist

MI & ischemia is termed according to the lead in which ST change , T inversion , Q wave present

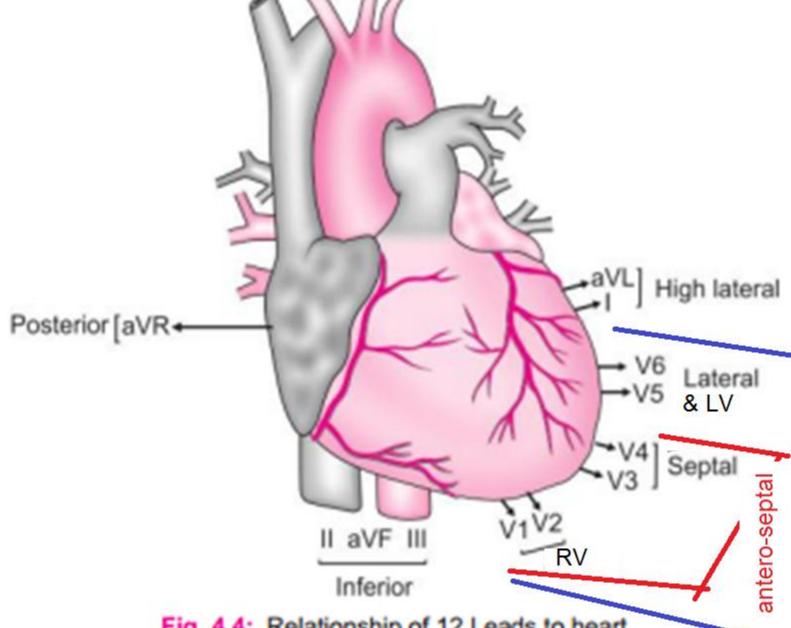
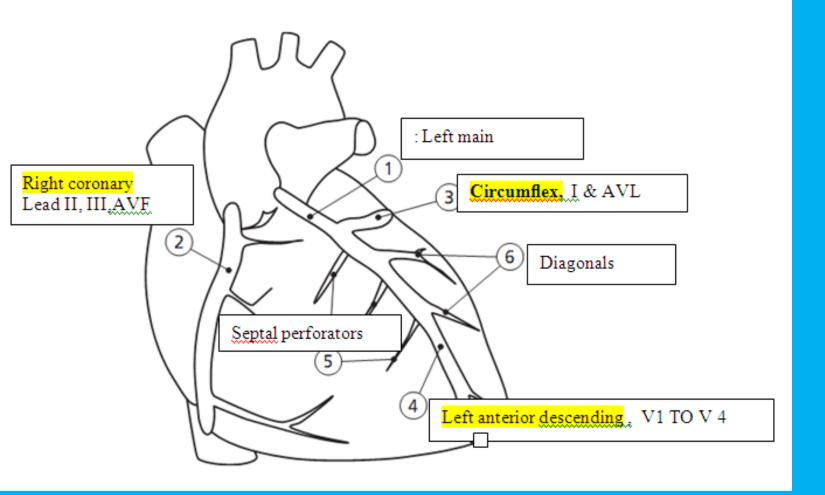
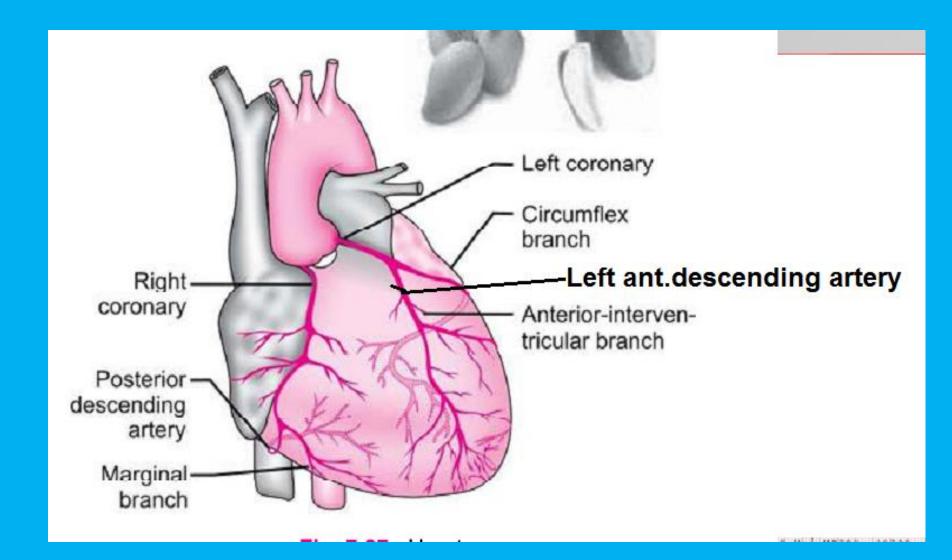


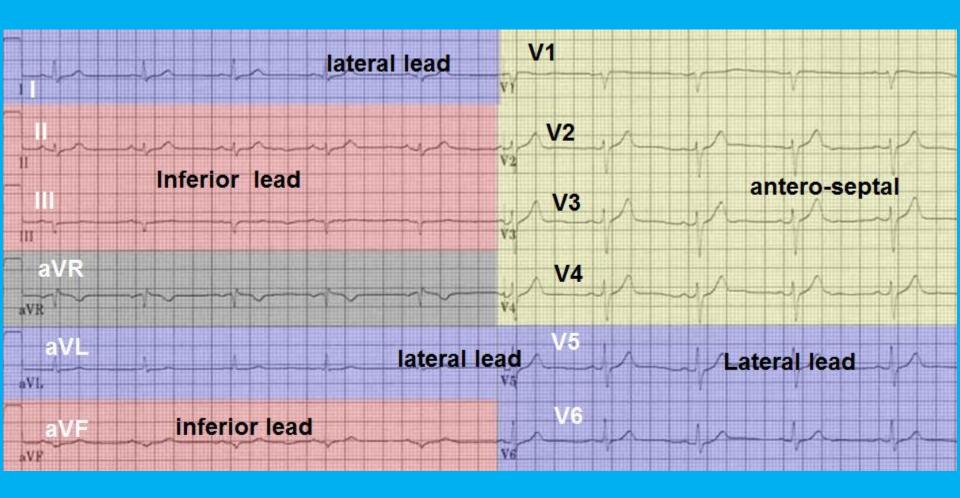
Fig. 4.4: Relationship of 12 Leads to heart

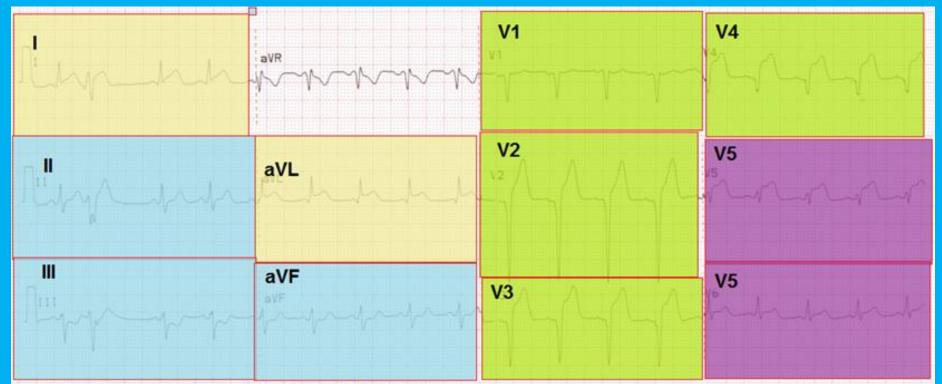


Acute ant.	Proximal left ant. Descending branch
Lateral	Diagonal br. Left ant. Des. Artery , left circumflex and marginal br. Left circumflex
Inferior	Right coronary artery

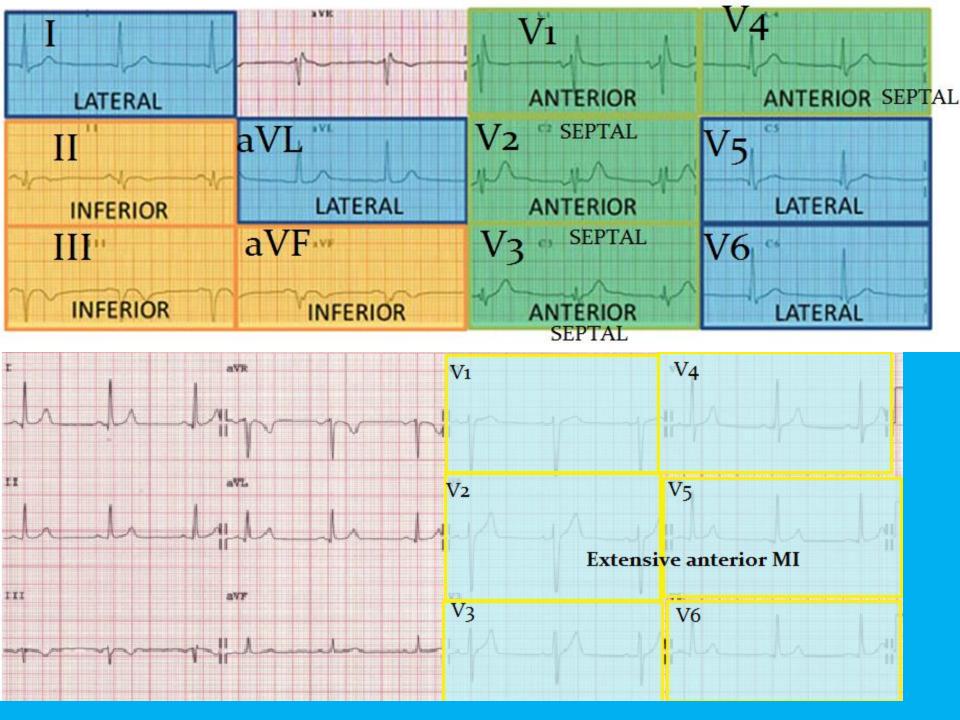


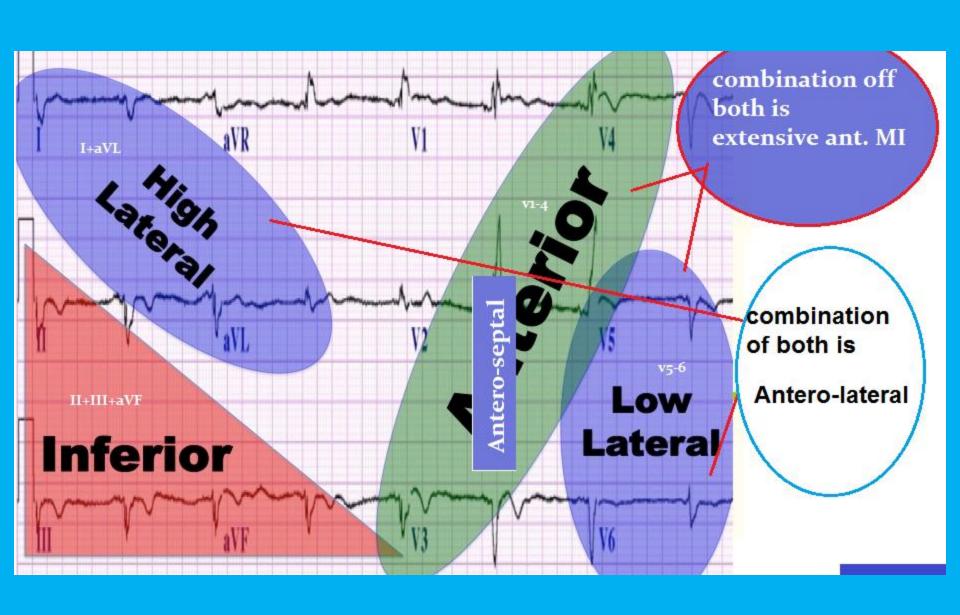
Localisation	ST elevation	Reciprocal ST depression	coronary artery	
Extensive Anterior MI	V1-V6	None	LAD	
Antero-Septal MI	V1-V4, disappearance of septum Q in leads V5,V6	none	LAD-septal branches	
Lateral MI	I, aVL, V5, V6	II,III, aVF	LCX or MO	
Antero-lateral	I, aVL, V1-6	II,III, aVF		
Inferior Mi	II, III, aVF	I, aVL	RCA (80%) or RCX (20%)	
Posterior MI	V7, V8, V9	high R in V1-V3 with ST depression V1-V3 > 2mm (mirror view)	RCX	
Right Ventricle MI	V1, V4R	I, aVL	RCA	
left anterior descending artery (LAD) and the ramus circumflexus (RCX). The right coronary artery (RCA)				





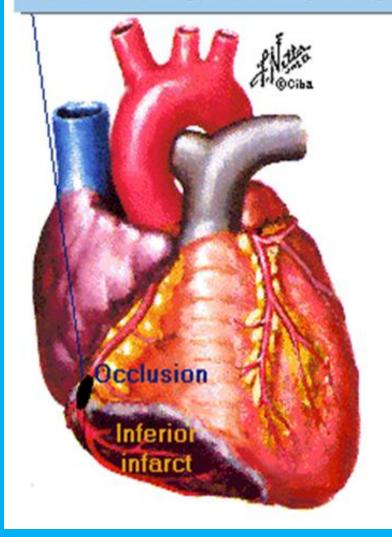
lateral lead = I,aVL,V5,V6 Anterolateral = I,aVL,V1-V6 inferior lead = II,III,aVF Antero-septal = V1-V4 extensive anterior = V1 to V6



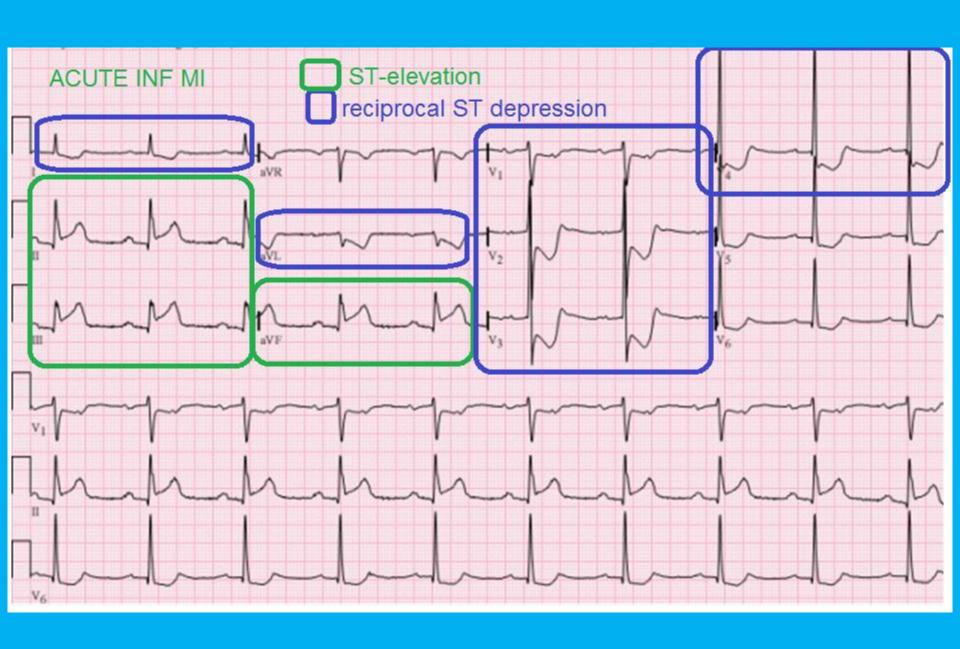


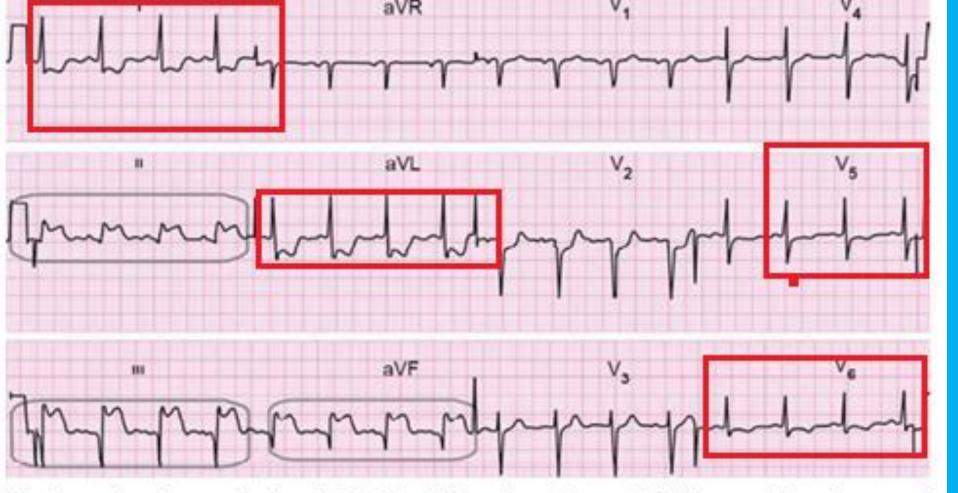


Occlusion of right coronary artery

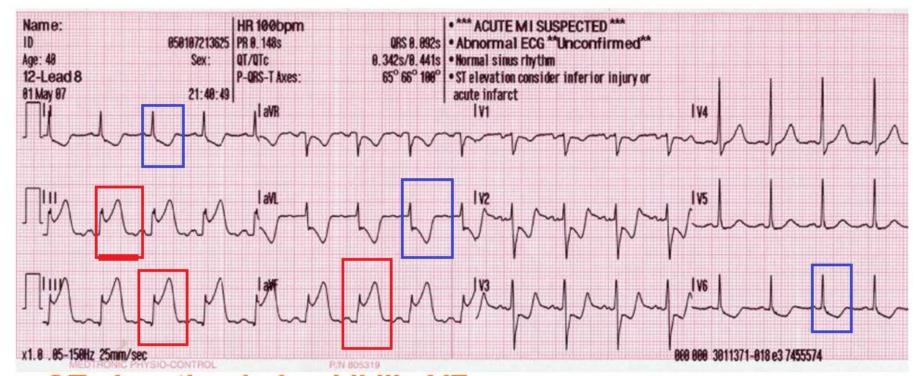


Inferior MI

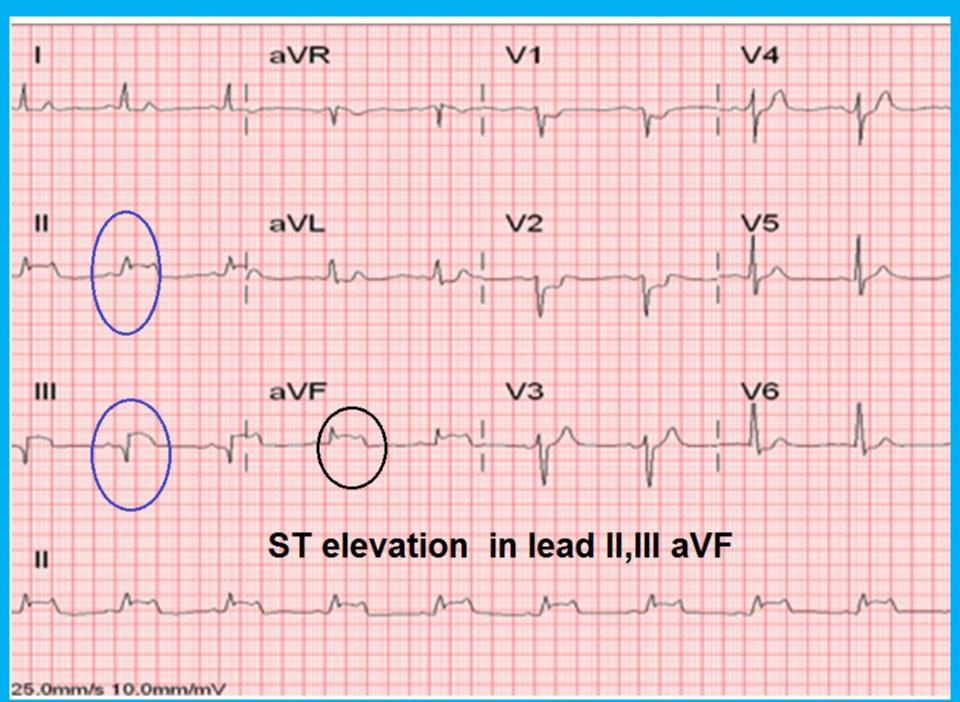


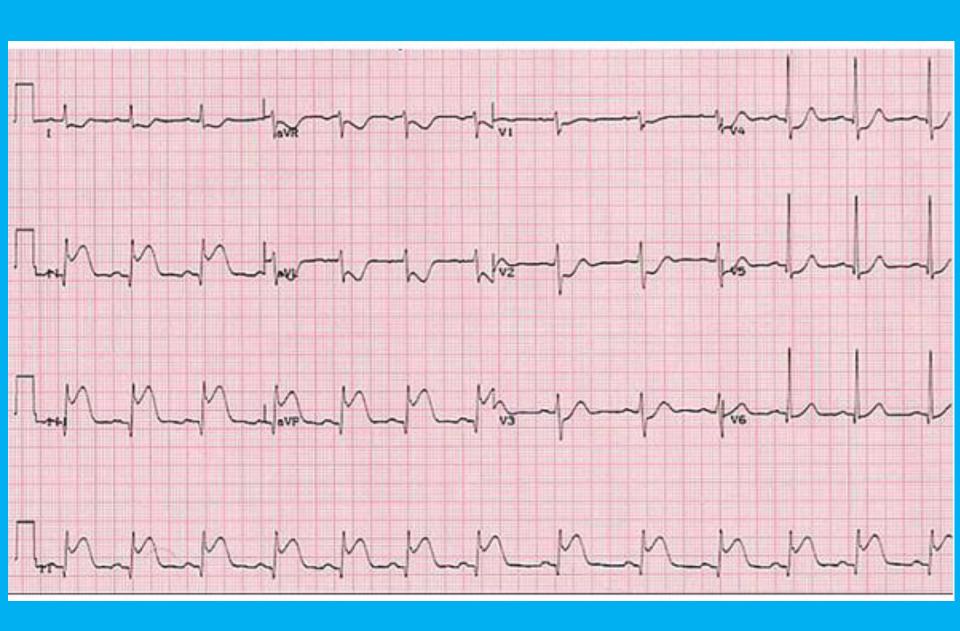


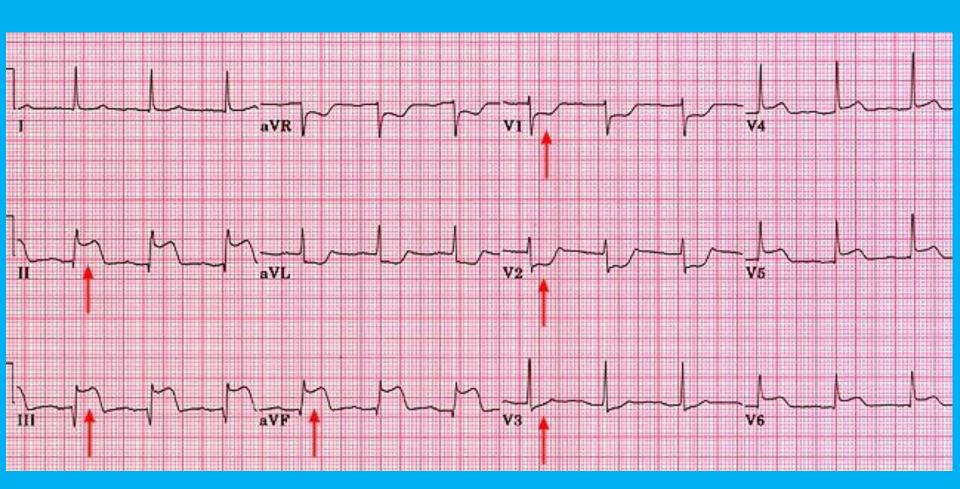
ST elevation is seen in leads II, III, aVF and reciprocal ST depression changes in I, aVL, V5, V6.



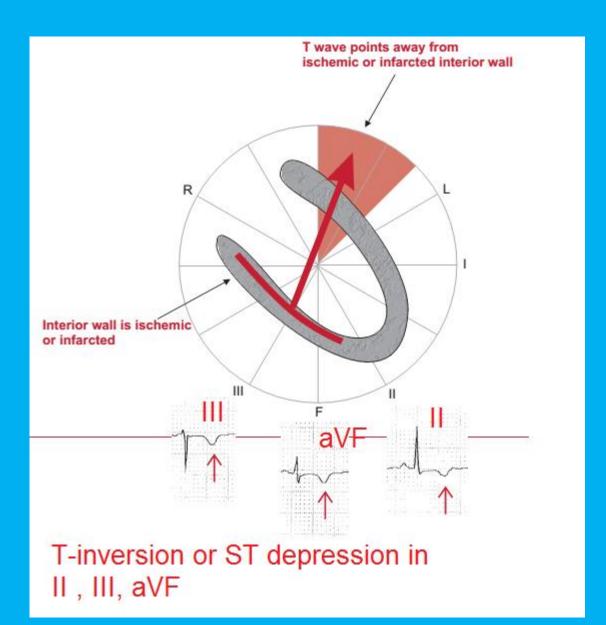
ST elevation in lead II,III,aVF reciprocal ST depression

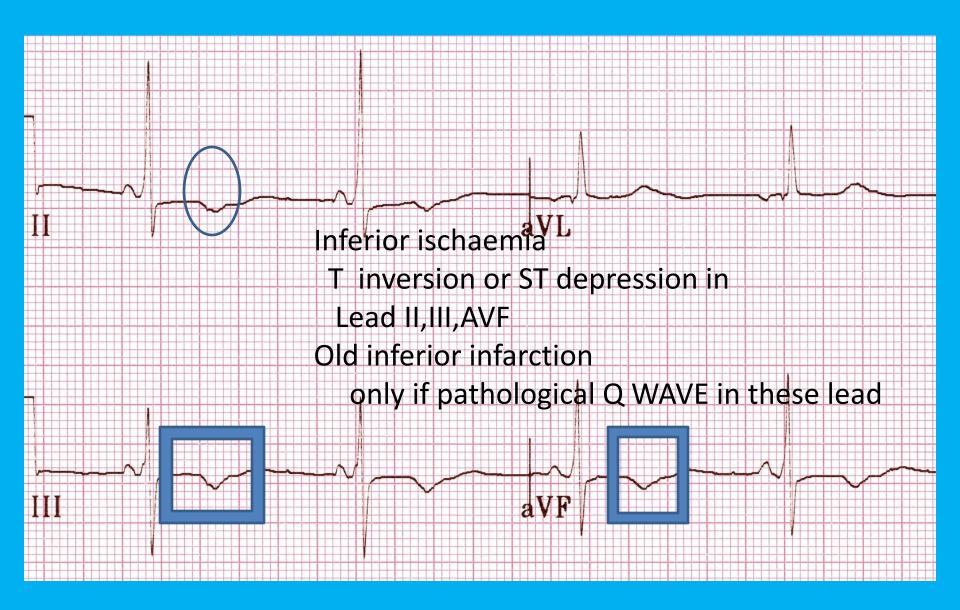


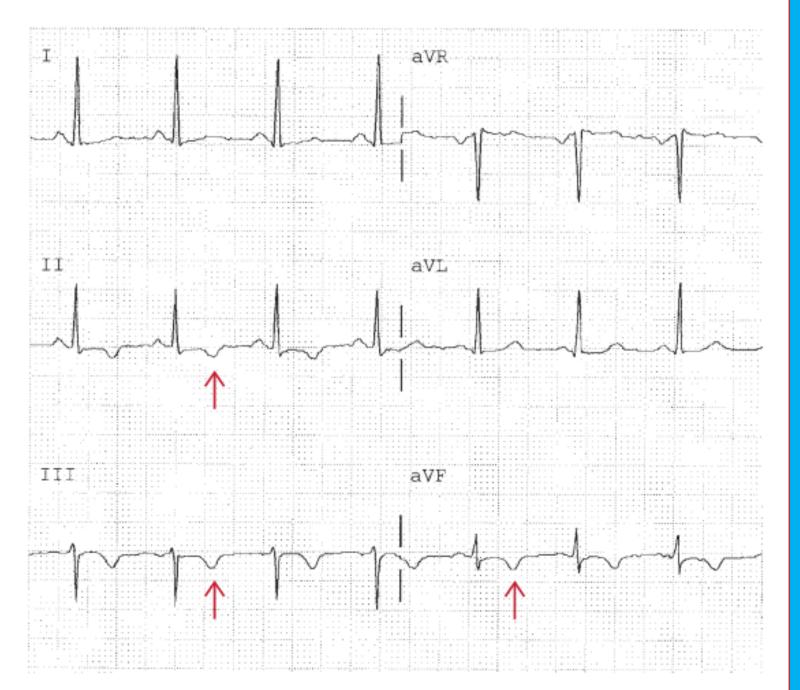


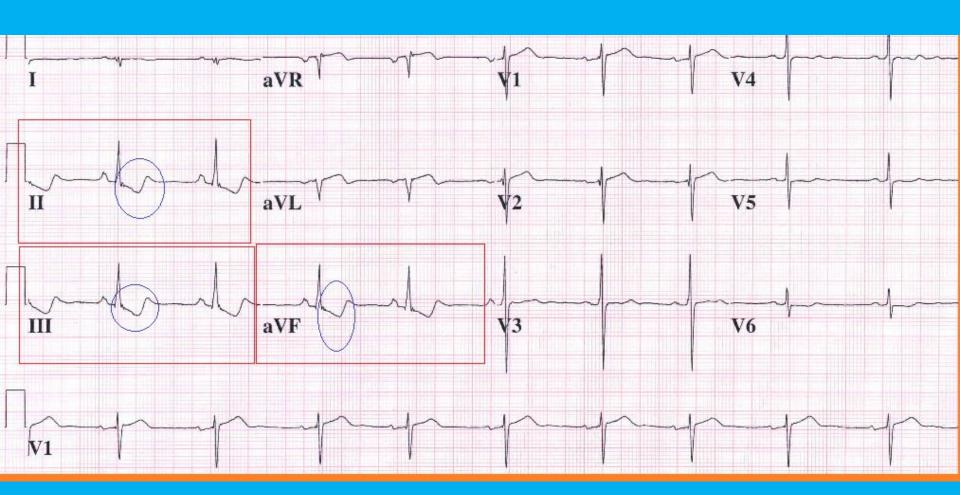










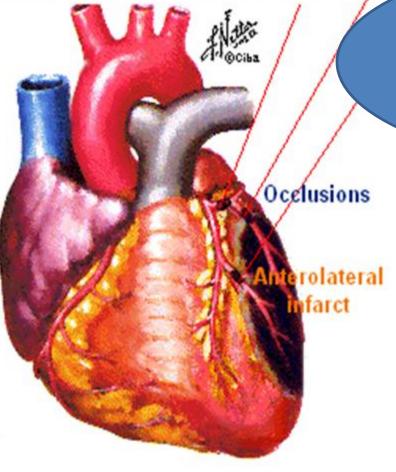


OCCLUSION OF Diagonal branch OR, marginal bra OR, left circumfl

Diagonal branch of left anterior descending artery

OR, marginal branch of left circumflex artery

OR, left circumflex coronary artery



Antero-lateral

Anterolateral wall MI:

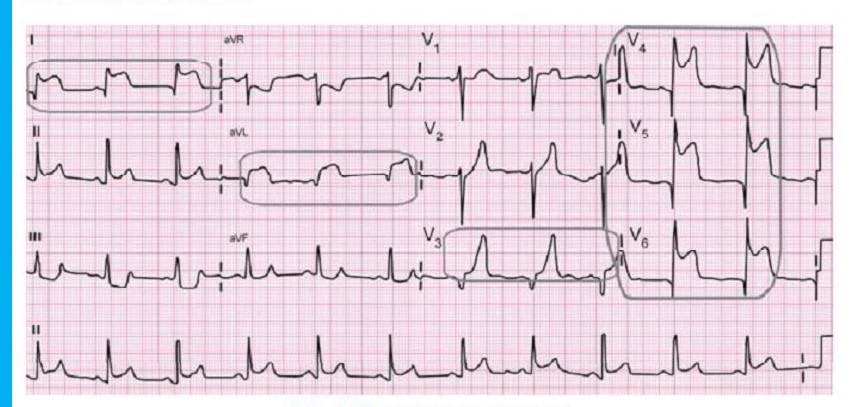
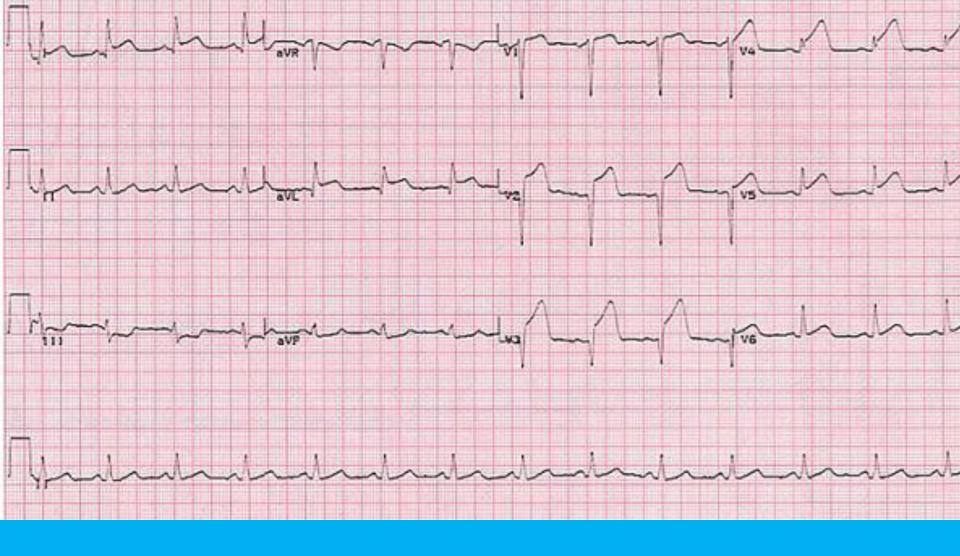
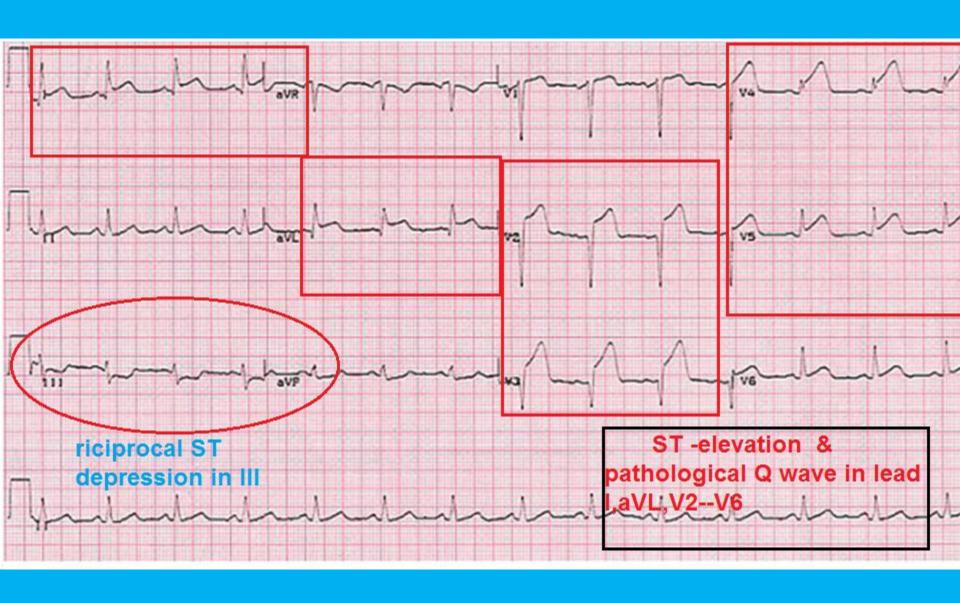


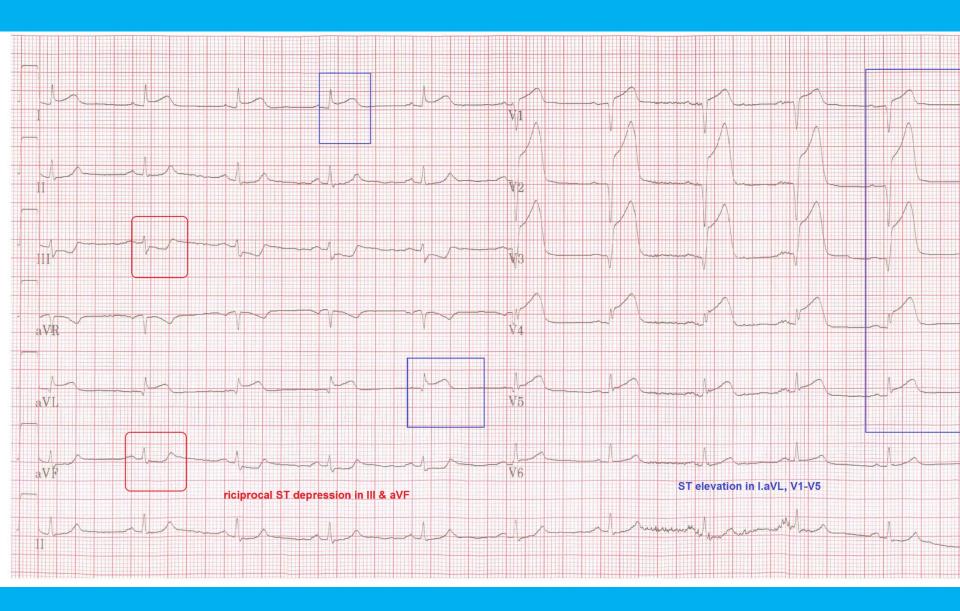
Fig. 7.24: Anterolateral wall MI

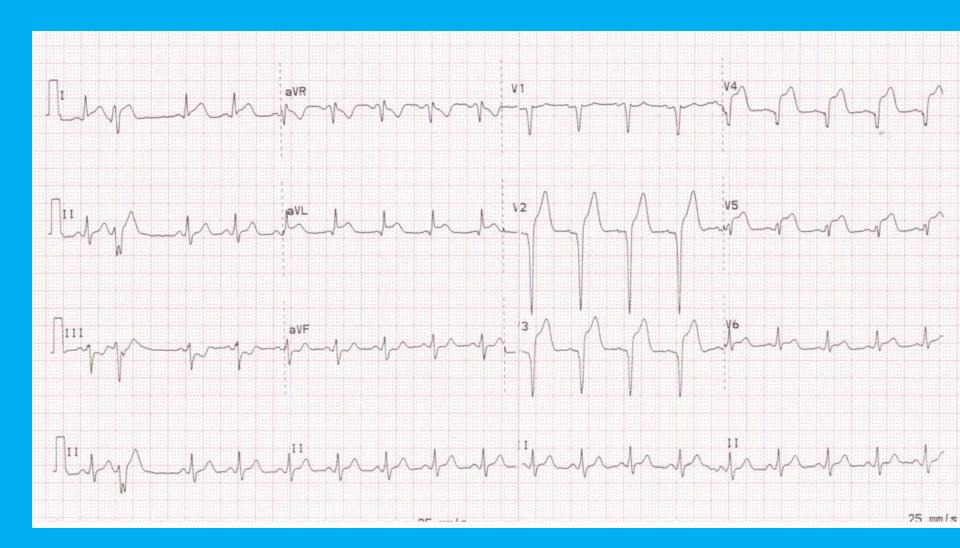
ST elevation is seen in leads I, aVL, V3, V4, V5, V6 and reciprocal ST depression in II, III, aVF.

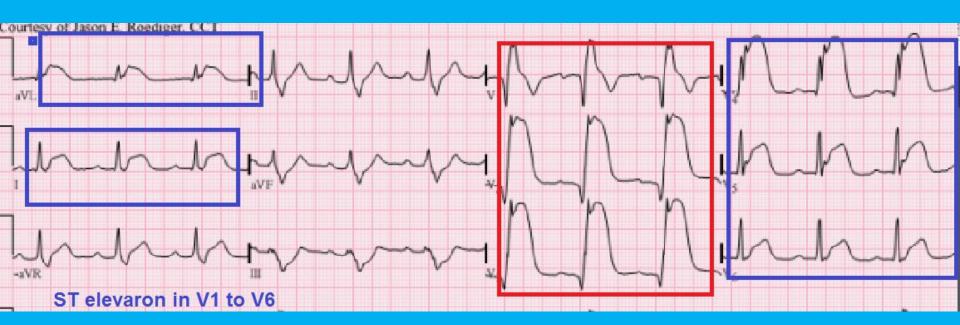


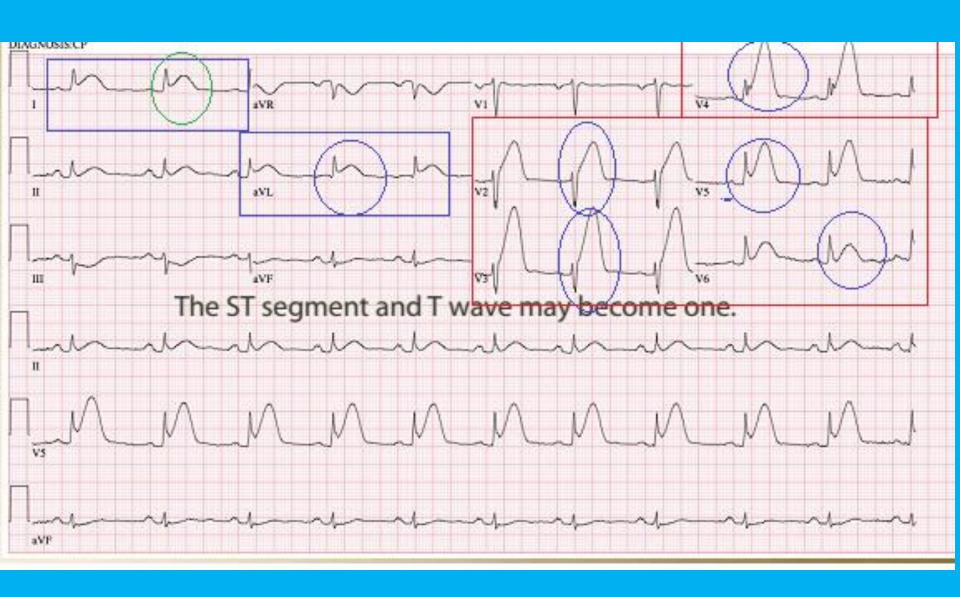
Anterolateral STEMI

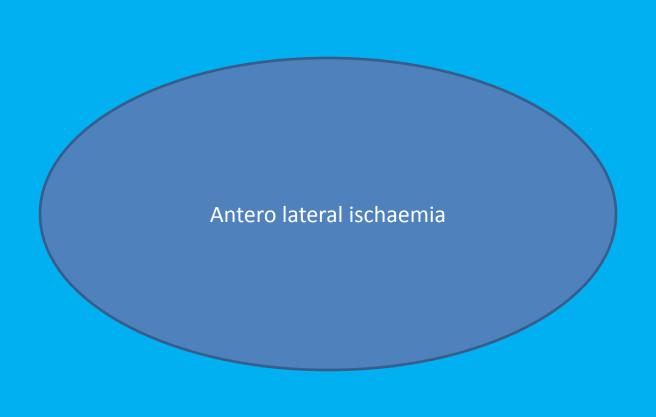


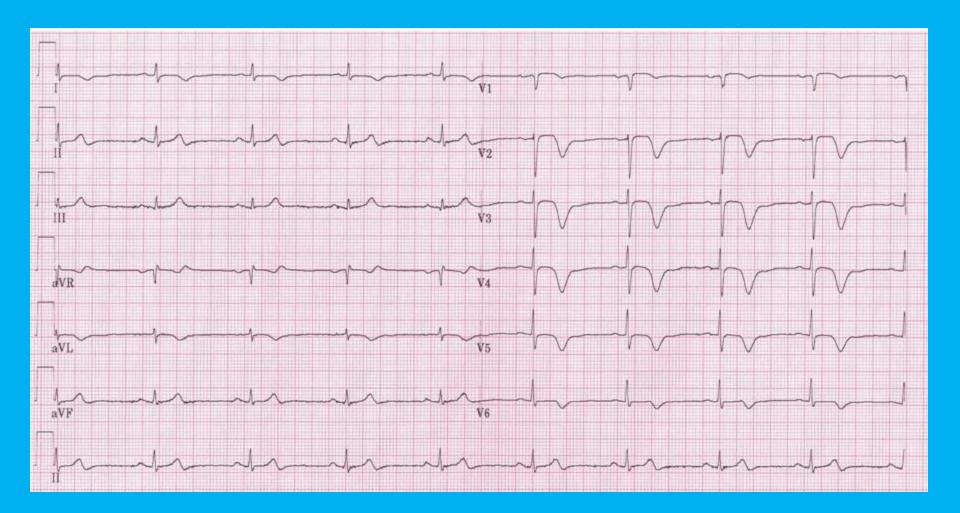


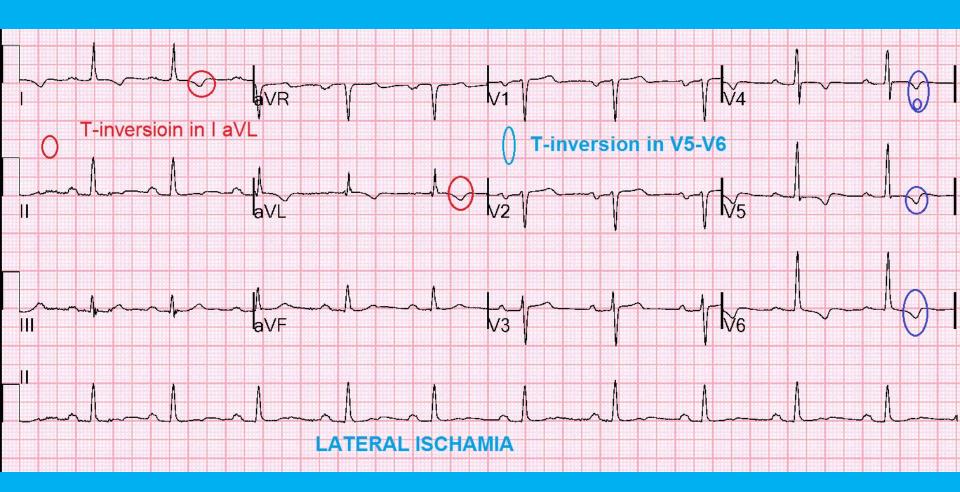


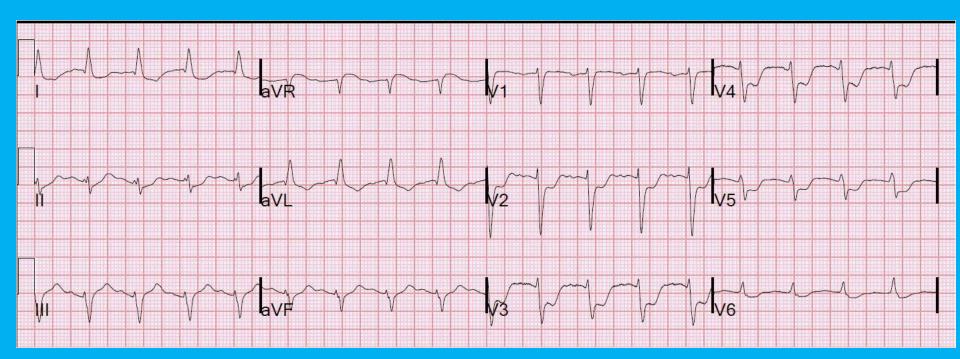




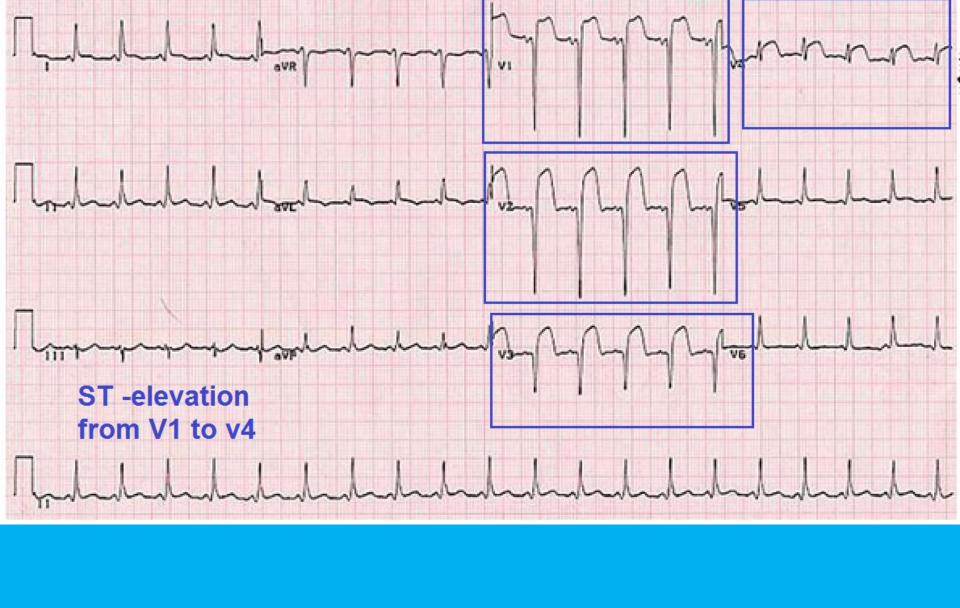


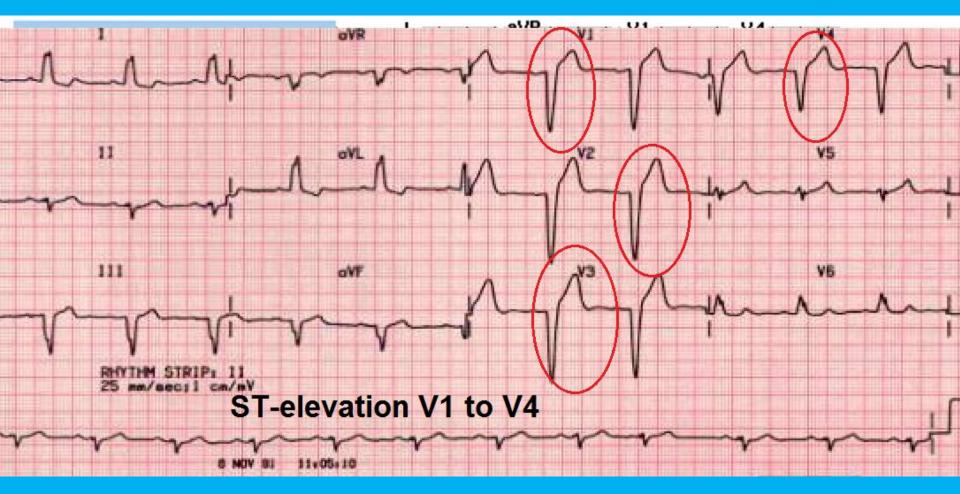


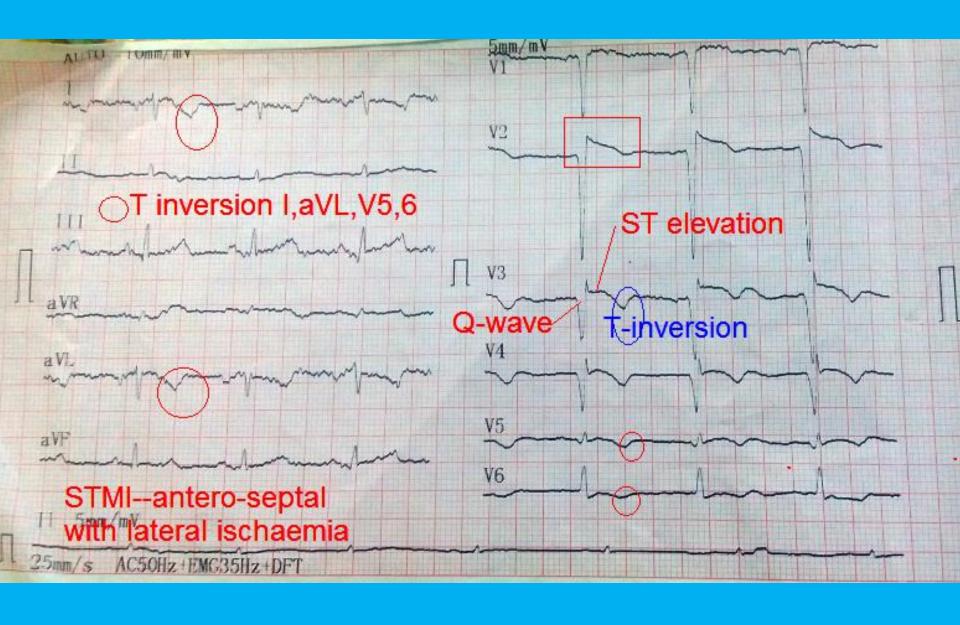


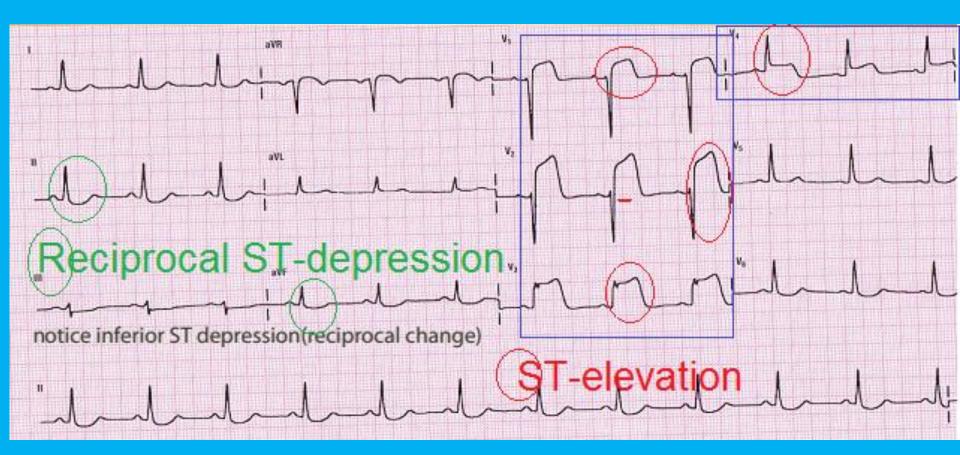




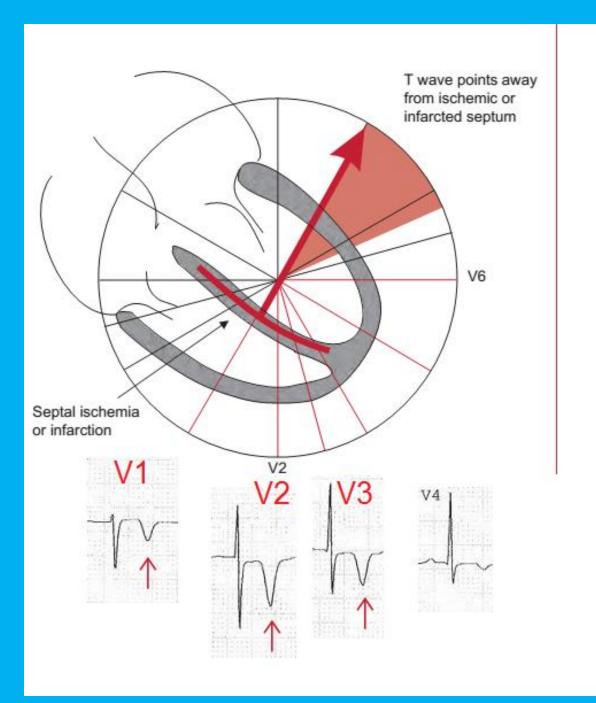


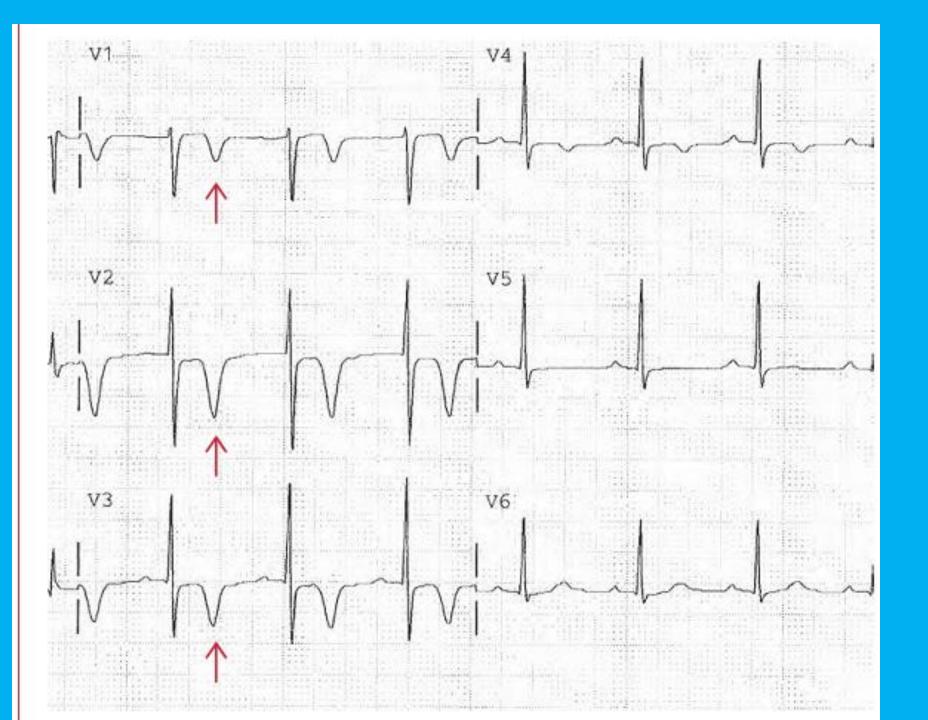


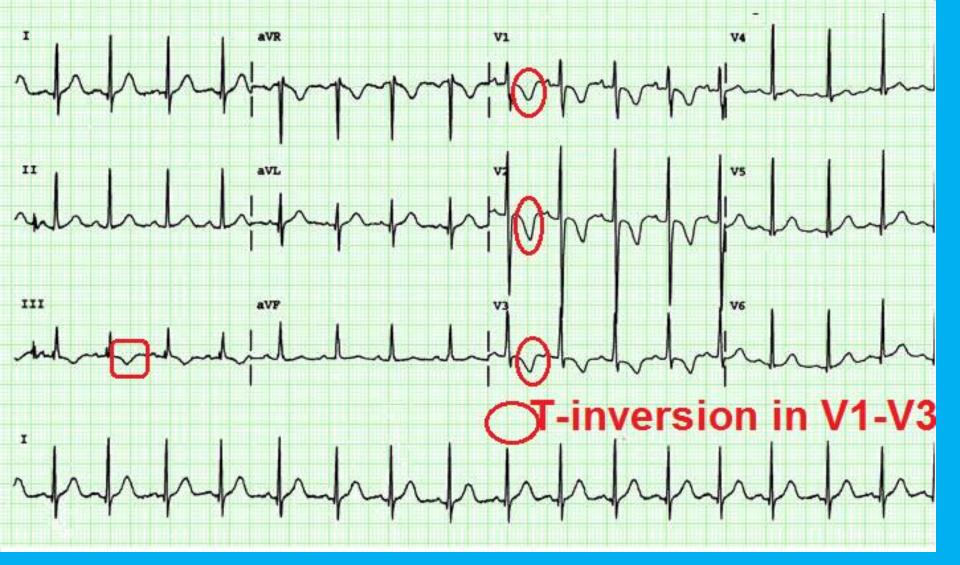


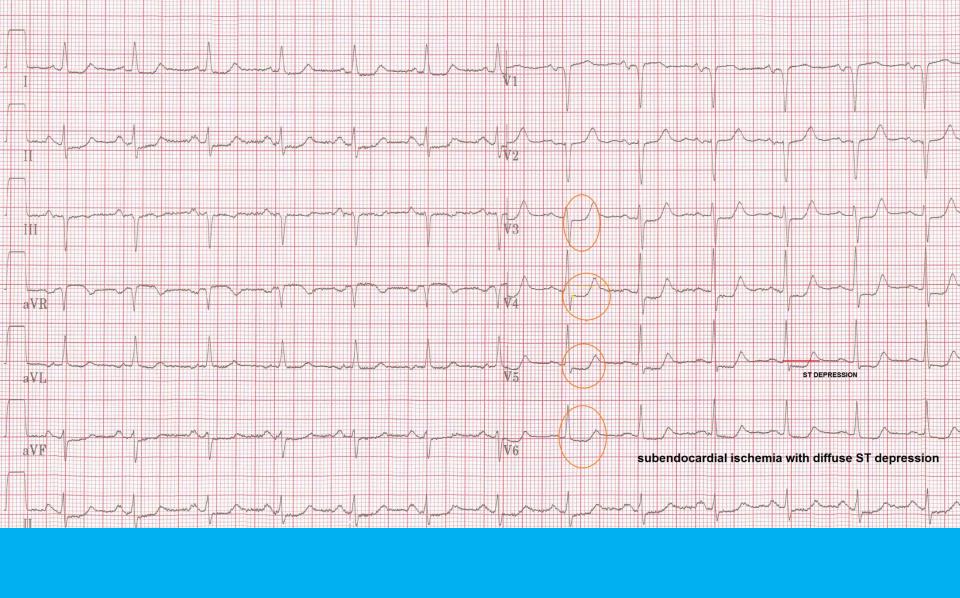


Antero-septal ischaemia

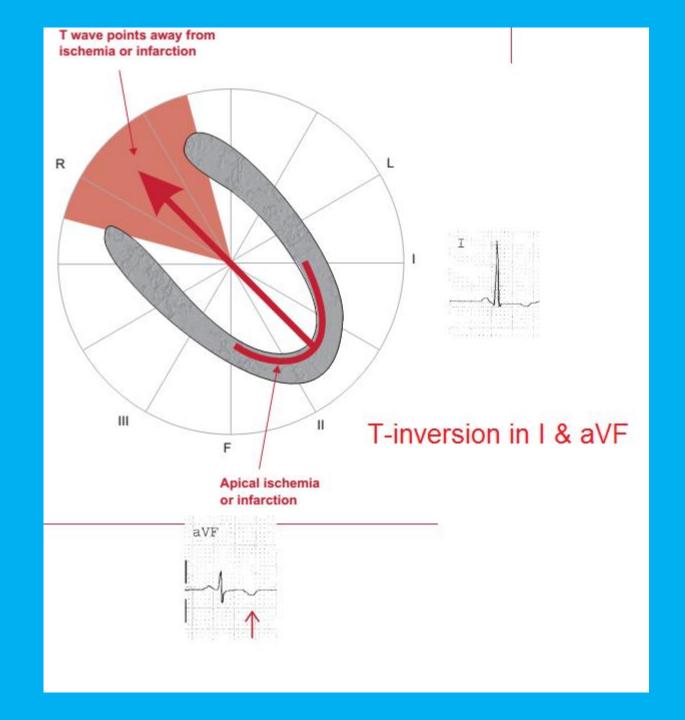


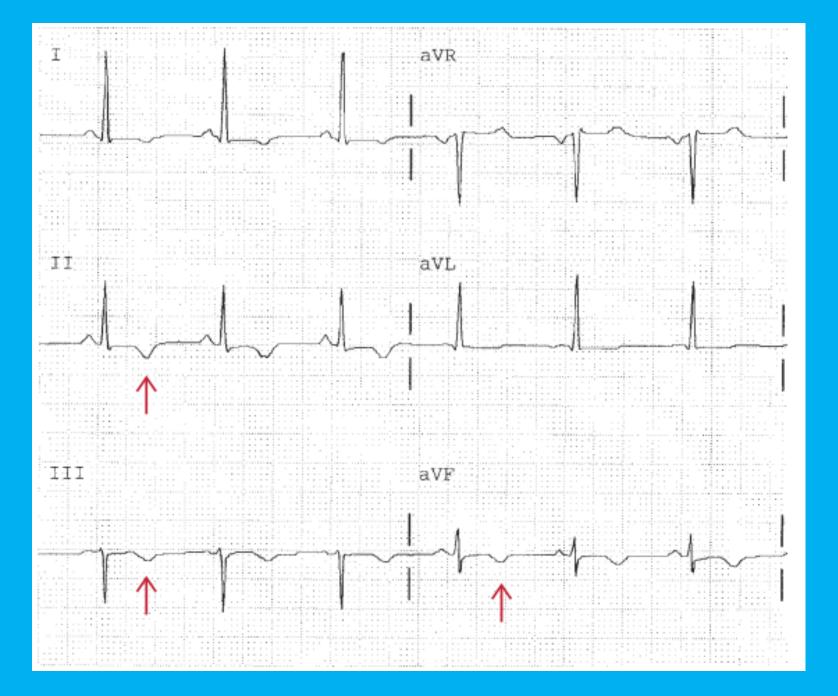






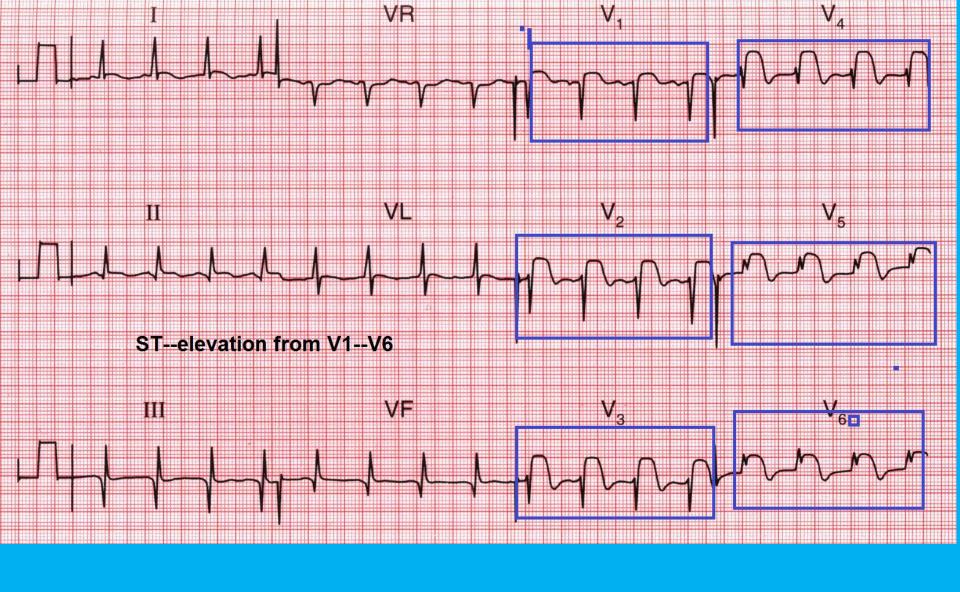
Apical ischaemia

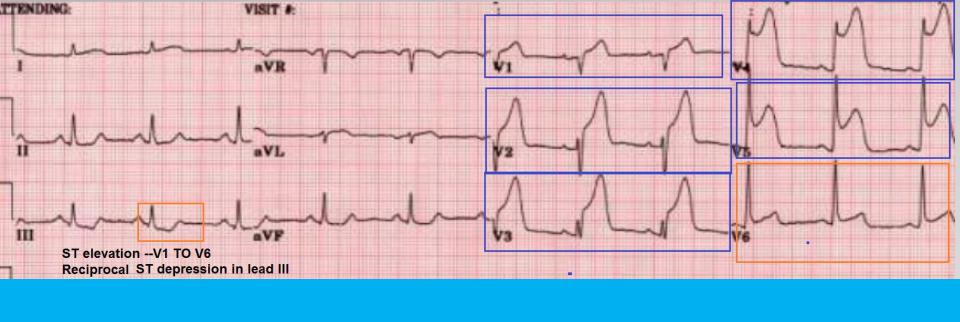


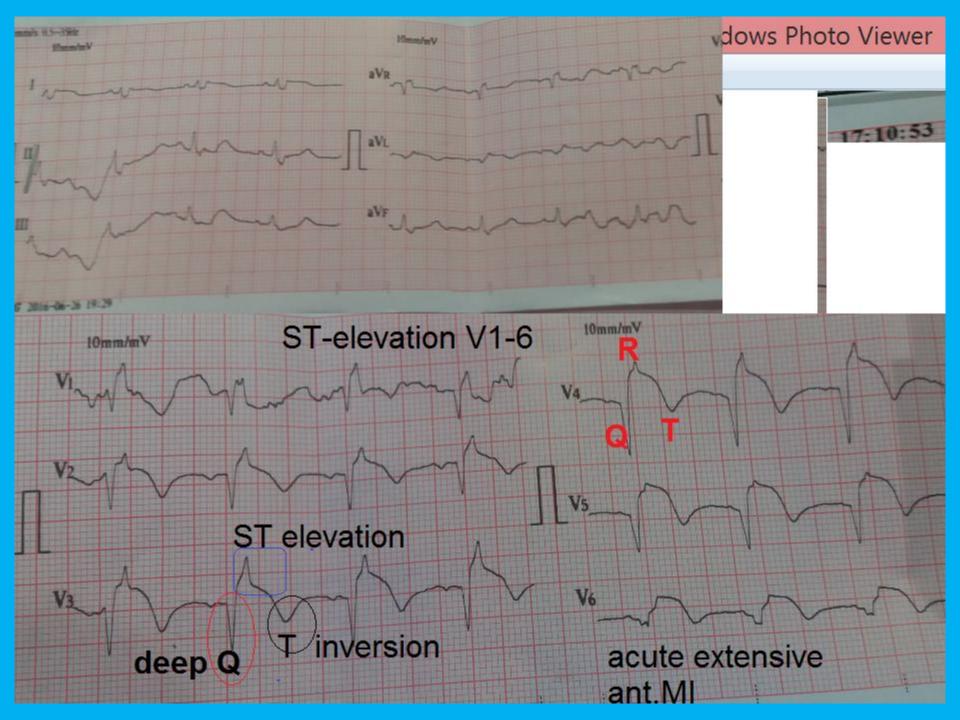


Extensive anterior Mi

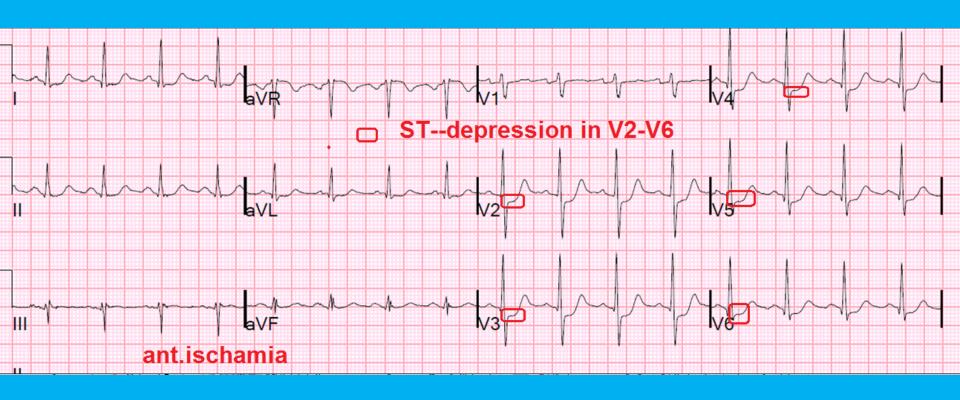
Anterior MI

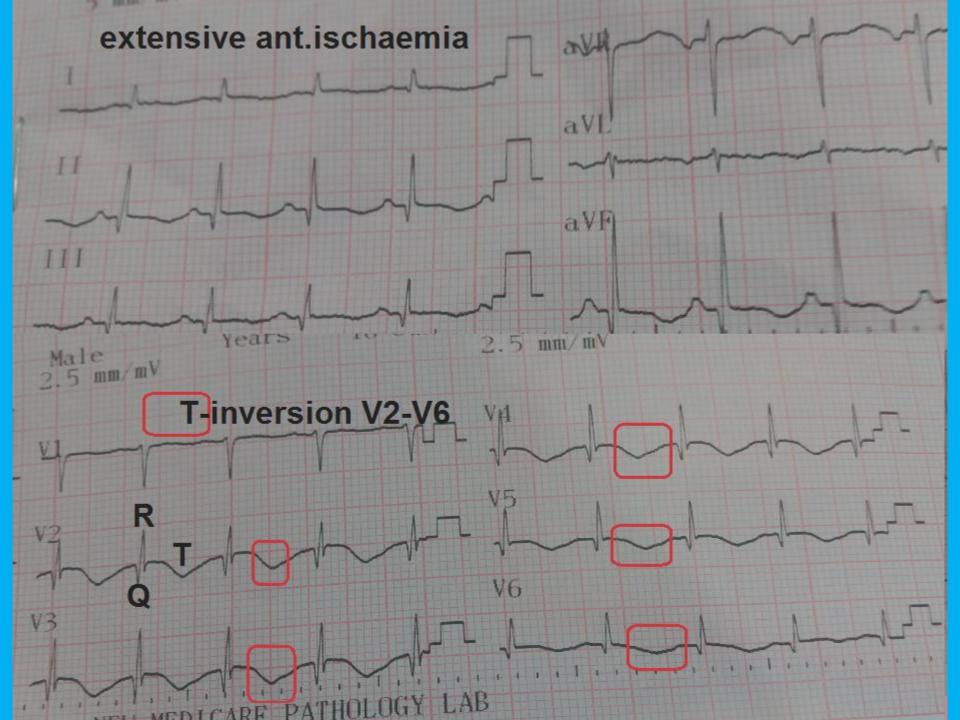


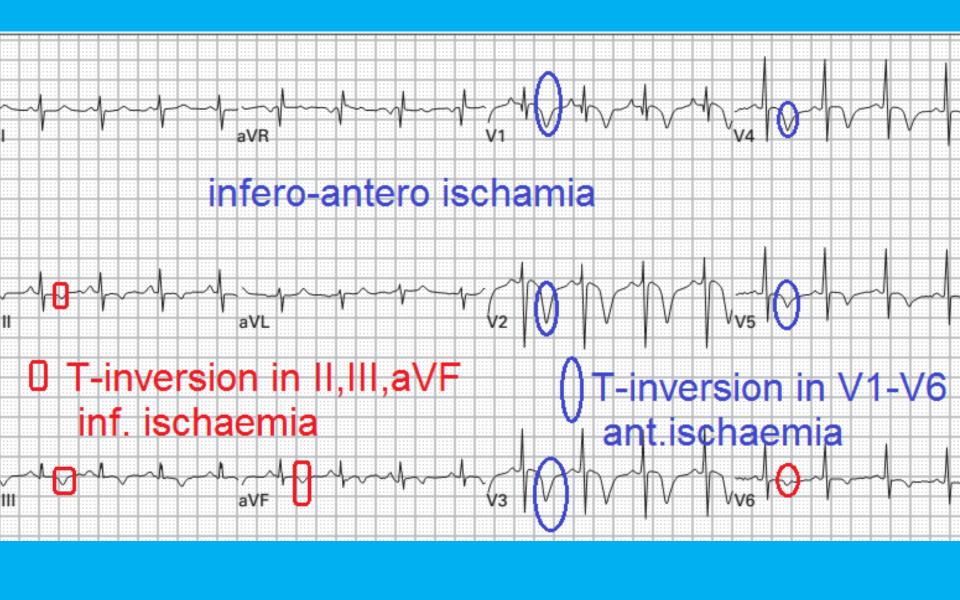




Anterior ischaemia







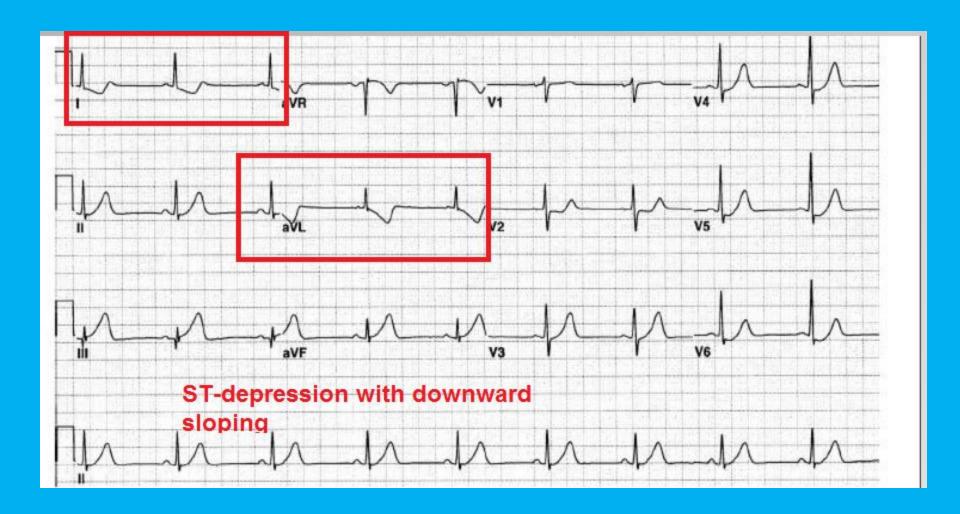
LATERAL MI

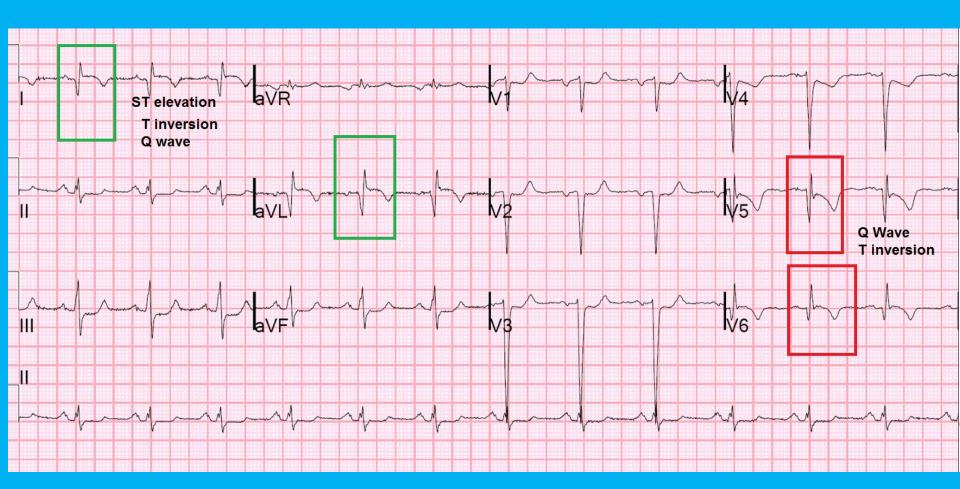
Section 6

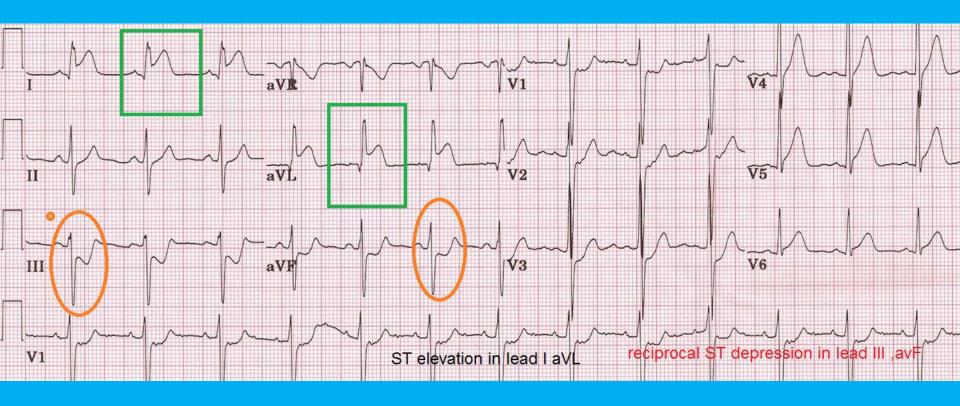
Localization of Myocardial Infarcts

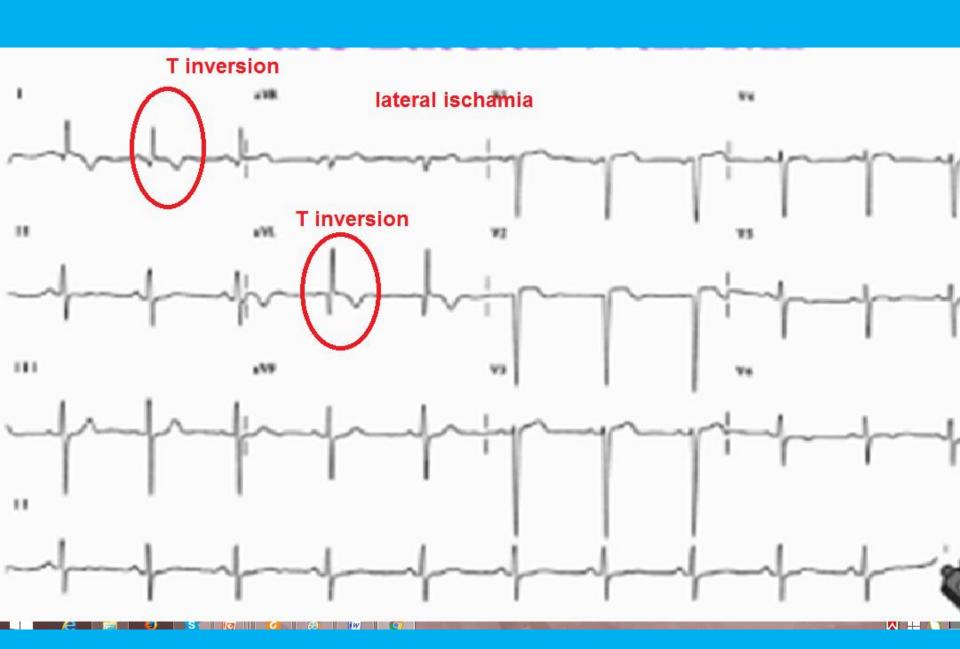
lateral MI

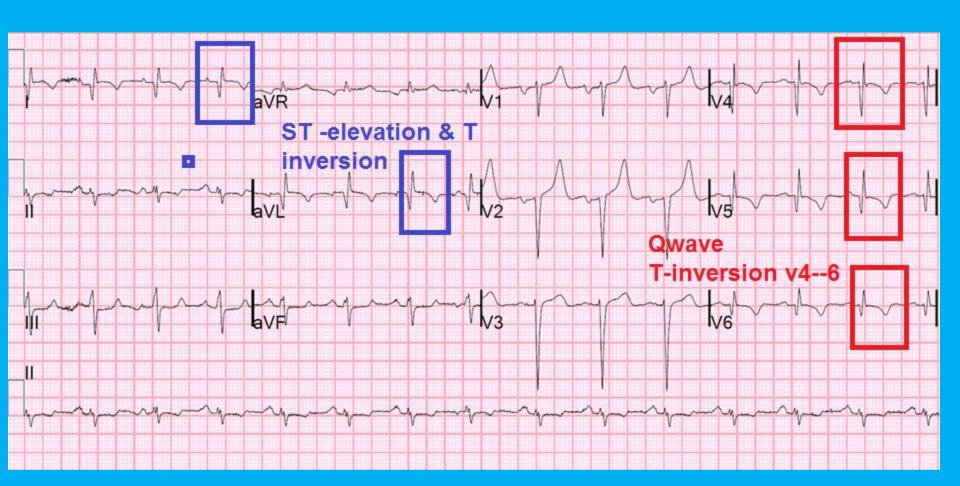
OCCLUSION OF Diagonal branch of left anterior descending artery OR, marginal branch of left circumflex artery OR, left circumflex coronary artery Ocelusions erolateral efarct Significant Q waves and T wave inversions in leads I, aVL, V5, and V6

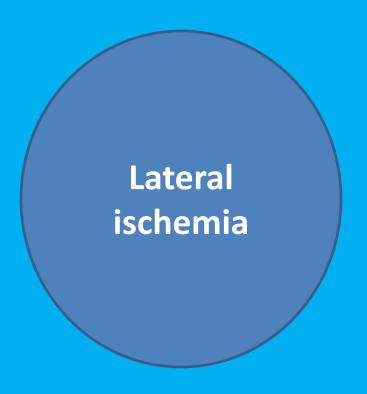


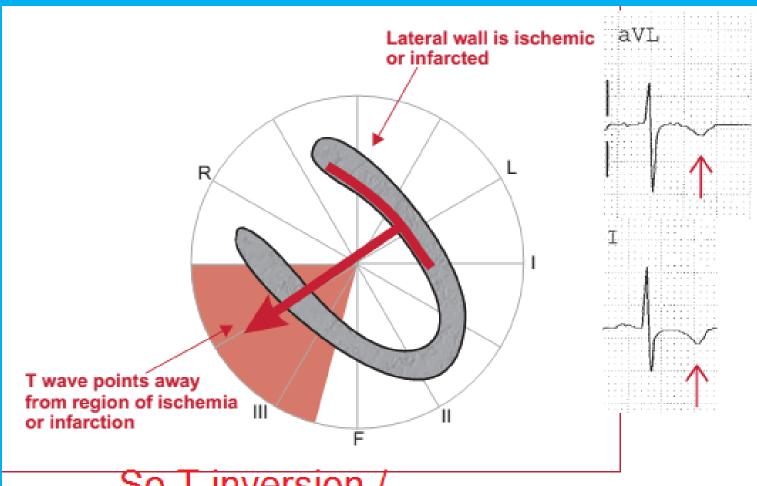




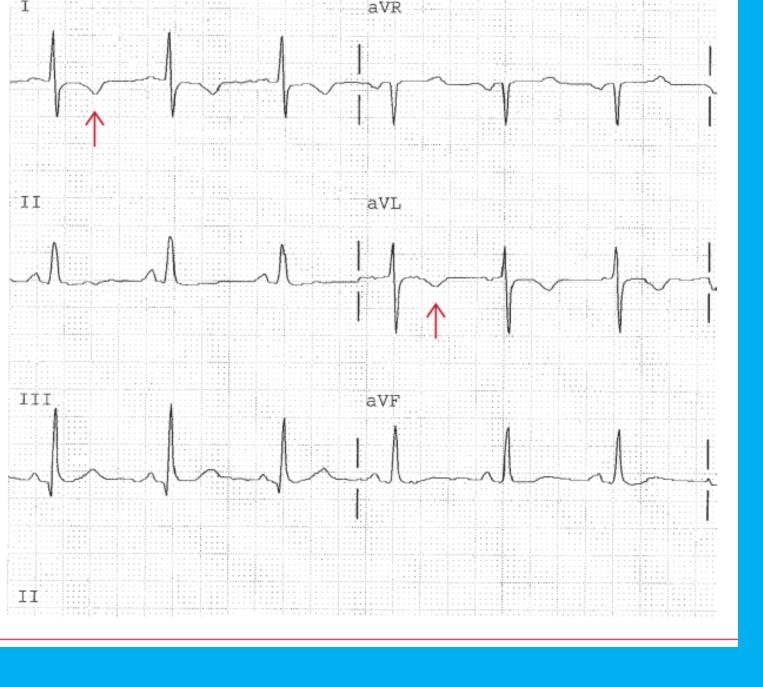




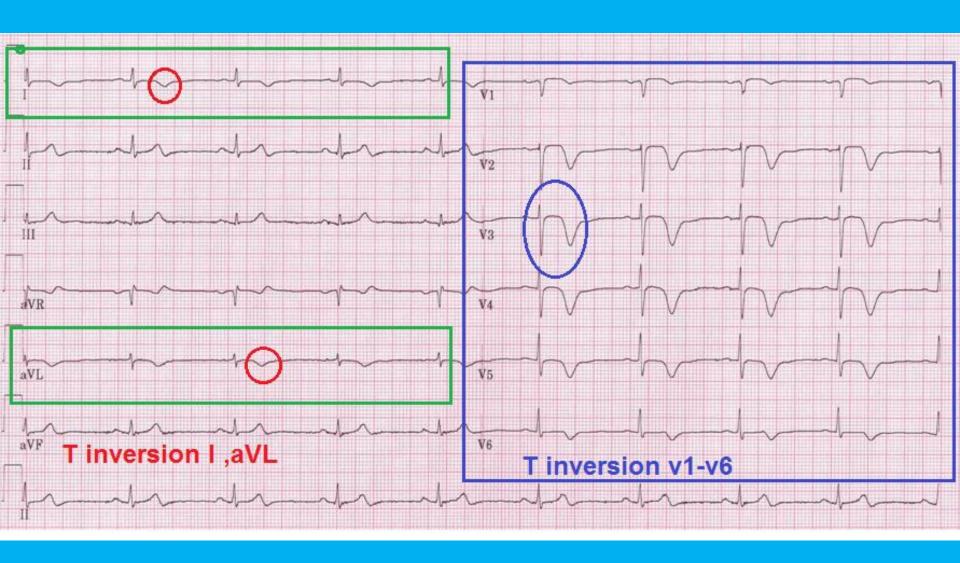


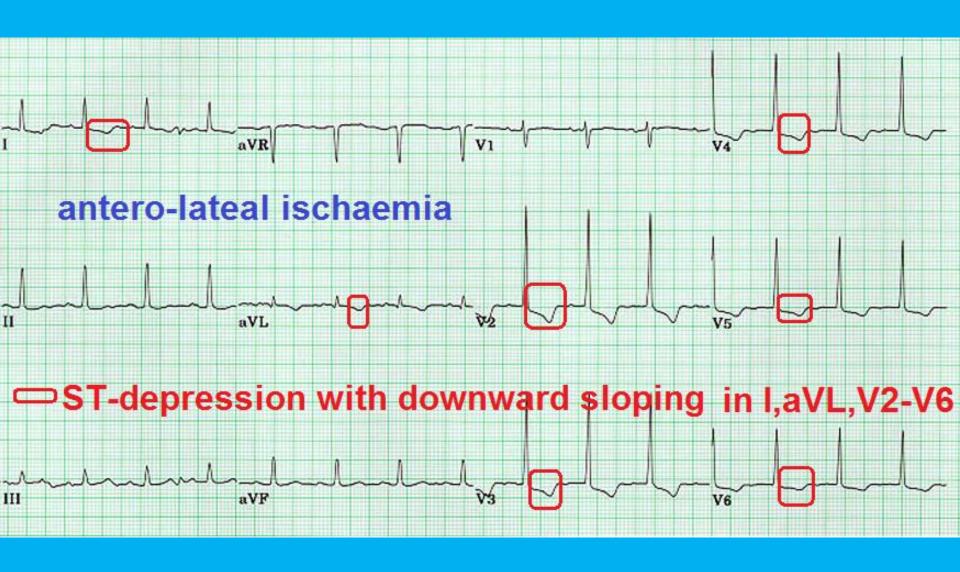


So T inversion / STdepression in I,aVL



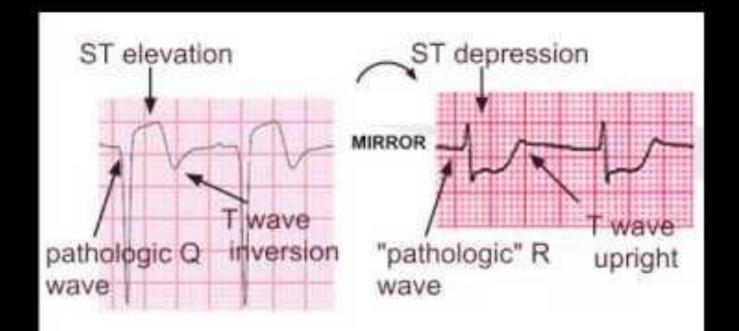
Antero –lateral ischaemia





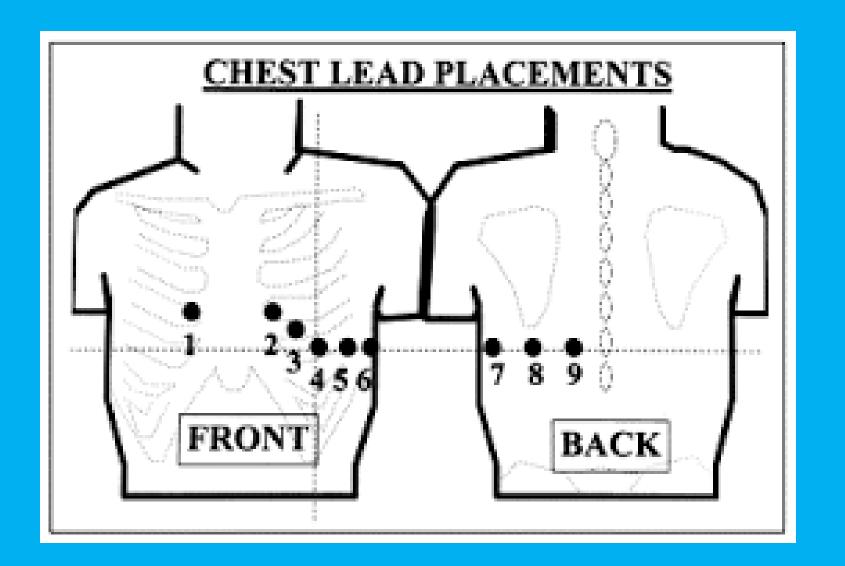
Posterior MI

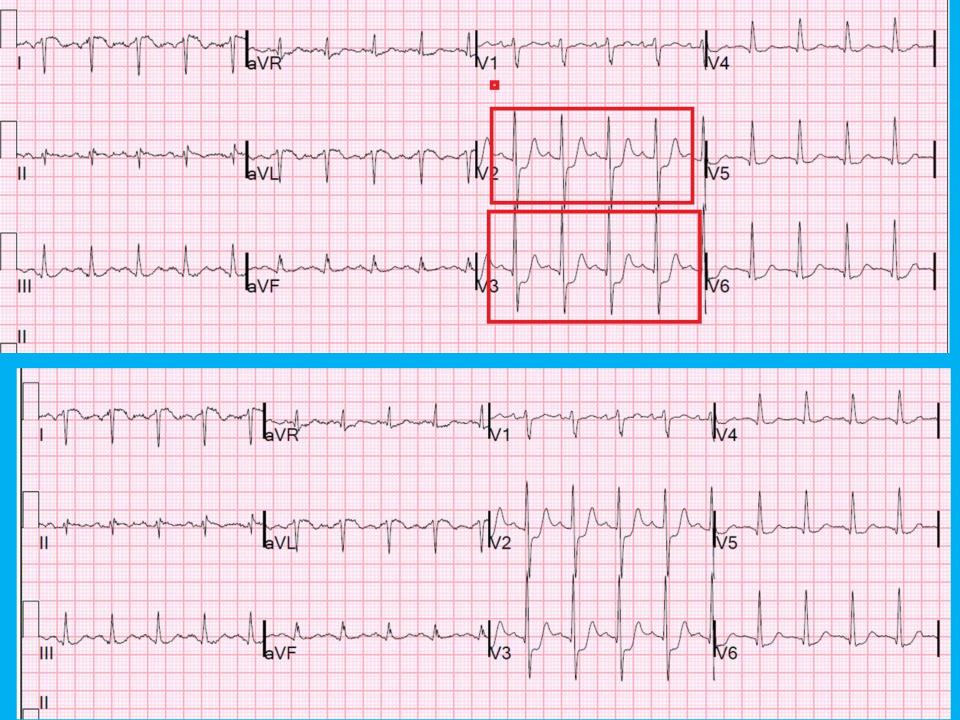




Posterior MI:

- ST depression
- "pathologic" R waves
- T waves upright





ST-segment depressions represent subendocardial or non-transmural ischemia. However, in the right precordial leads (V1-V3) your differential for ST-segment depression should include right ventricular hypertrophy with strain and posterior STEMI in addition to anterior subendocardial ischemia. Similarly, the differential for tall R-waves in leads V1-V3 should include the possibility that they represent posterior Q-waves or that upright T-waves may actually be posterior T-wave inversions. It is important to rethink the approach to the ECG and to maintain a high index of suspicion for PMI when the following findings are seen on the standard ECG:

- 1. ST-segment depression (horizontal >> downsloping or upsloping) in leads V1-V3
- 2. Prominent upright T-waves in leads V1-V3
- 3. Combination of horizontal ST-segment depression with
- 4. Prominent R-waves in leads V1-V3
- 5. R/S ratio > 1 in lead V2
- 6. Co-existing acute inferior and/or lateral myocardial infarction

that subendocardial ischemia DOES NOT LOCALIZE on the ECG, and usually is in leads II, III, aVF and V4-V6. But, again, this does not tell you which artery is involved. **Second**, ST depression in V1-V3, vs. V4-V6, is much more likely to be posterior than subendocardial ischemia. **Third**, patients at higher risk of NSTEMI (older, more risk factors, h/o angiogram with multivessel disease) are much more likely to have subendocardial disease (vs. younger smoker). **Fourth**, patients with reasons to have demand ischemia (tachycardia, sepsis, GI Bleed, etc.) are much more likely to have subendocardial ischemia (like in a stress test); those with posterior MI are much more likely to present with onset of chest pain and with normal vital signs. **<u>Fifth</u>**, look for tall R-waves in V1-V3 (the analog of Q-waves in other locations). <u>Sixth</u>, placement of posterior leads (take leads V4-V6 and place them at the level of the tip of the scapula, with V4 placed at the posterior axillary line ("V7"), V6 at paraspinal area ("V9"),

<u>First</u>, you should know that when there is precordial ST depression due to subendocardial

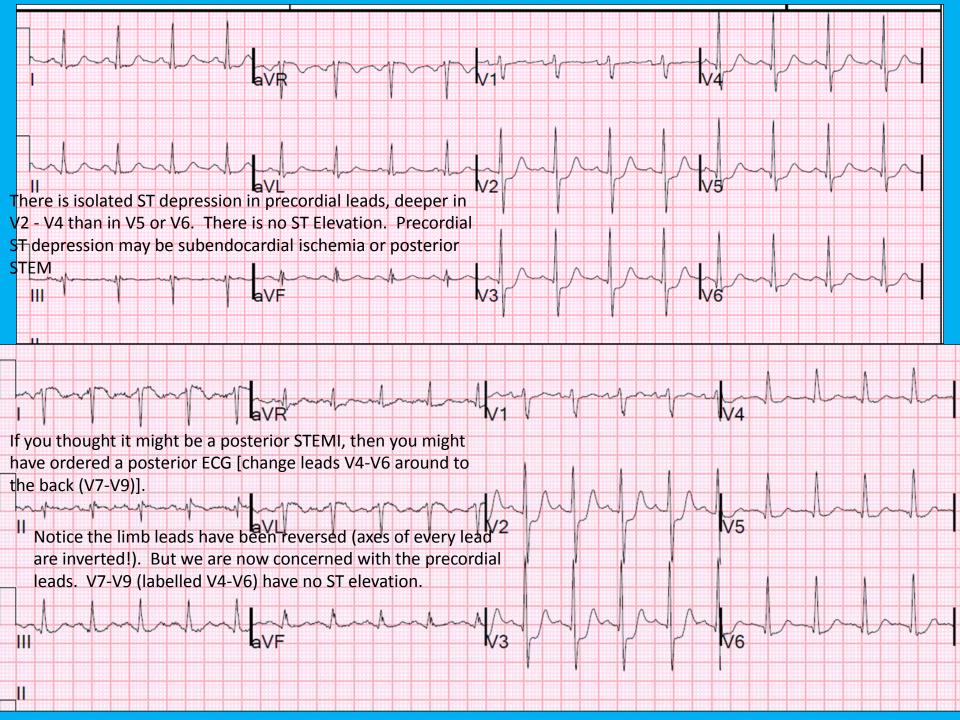
ischemia, it is not necessarily due to anterior wall ischemia. Data from stress testing shows

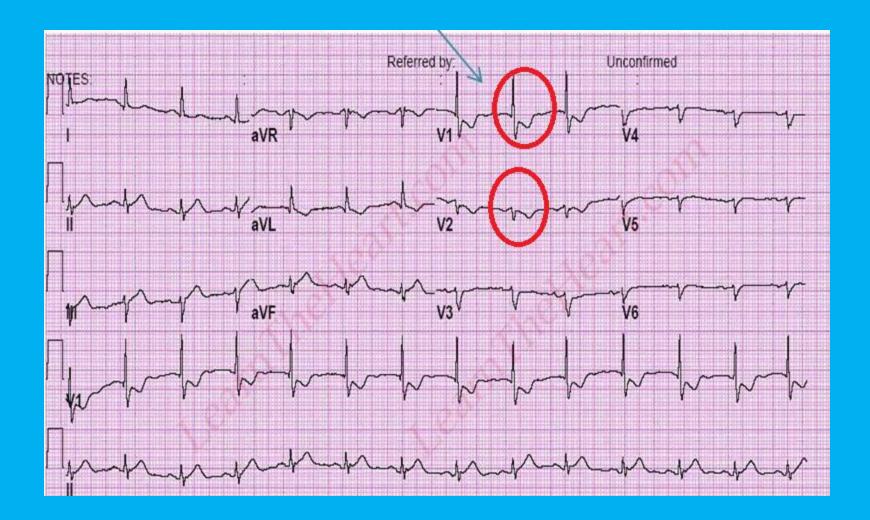
<u>Seventh</u>, an <u>immediate echocardiogram</u> can make the distinction. These are very difficult and it is very hard to detect a posterior wall motion abnormality unless you are very experienced. I recommend a formal study with Definity before concluding there is no posterior wall motion abnormality.

and V5 ("V8") between them. At lease 0.5 mm of ST elevation in just one lead is very

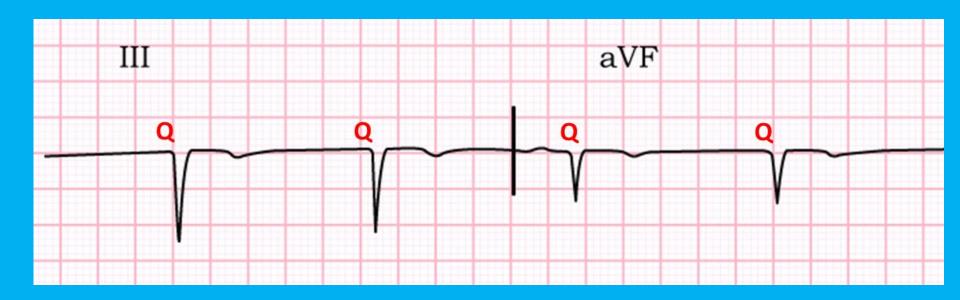
sensitive and specific for posterior MI.

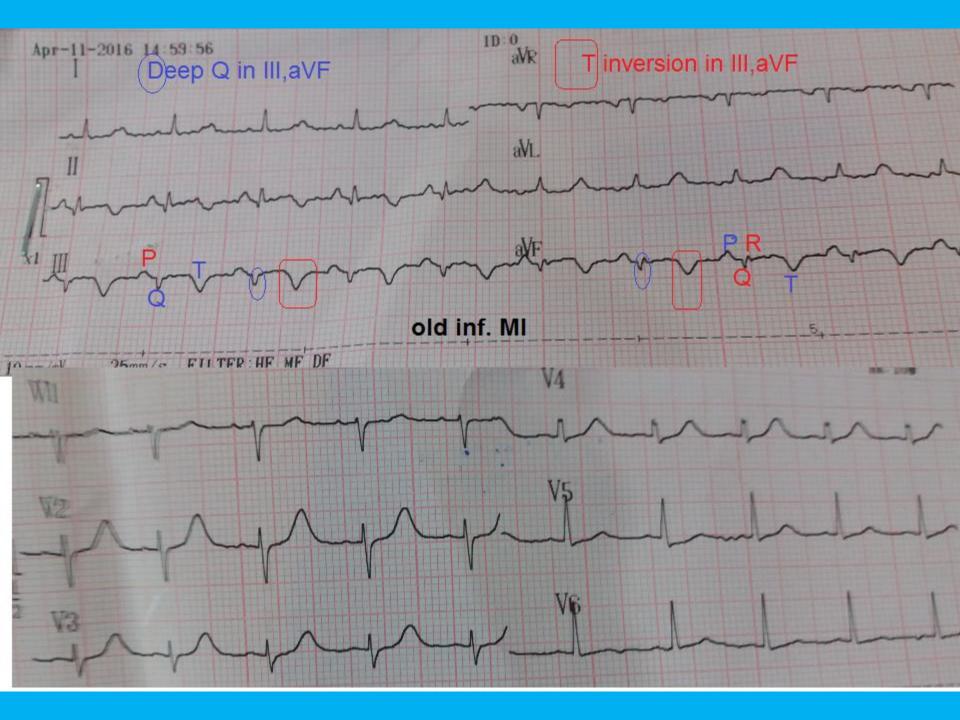
<u>Eighth</u>, see above. Whether or not it is STEMI, the cath lab should be activated if the ischemia cannot be controlled medically: aspirin, nitro, beta blockers, clopidogrel, heparin/enoxaparin, GP IIb/IIIa inhibitor.

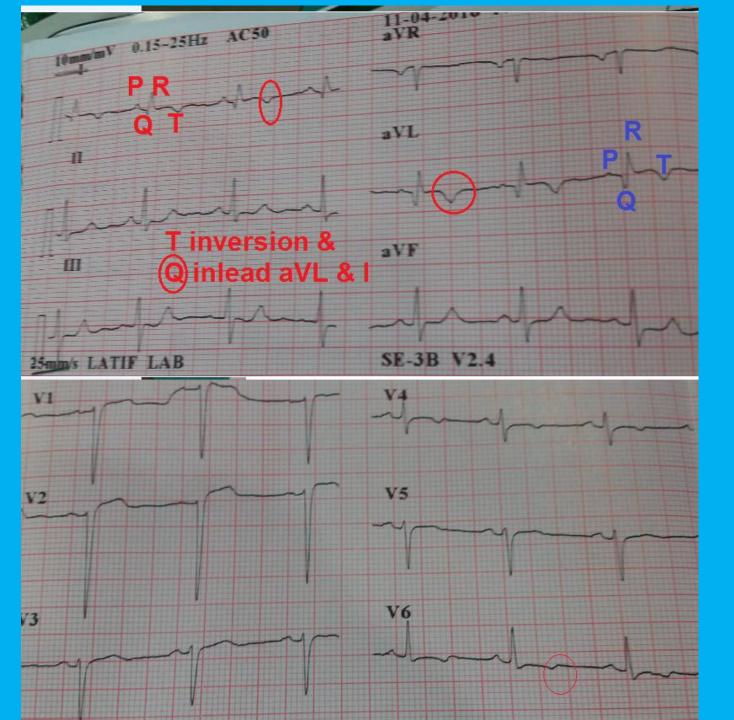


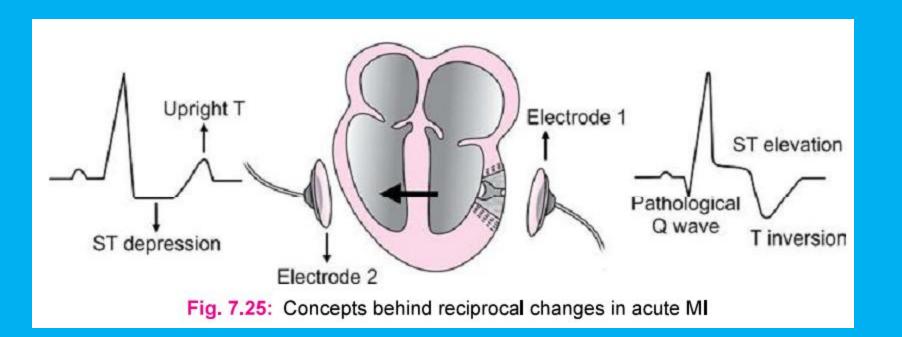








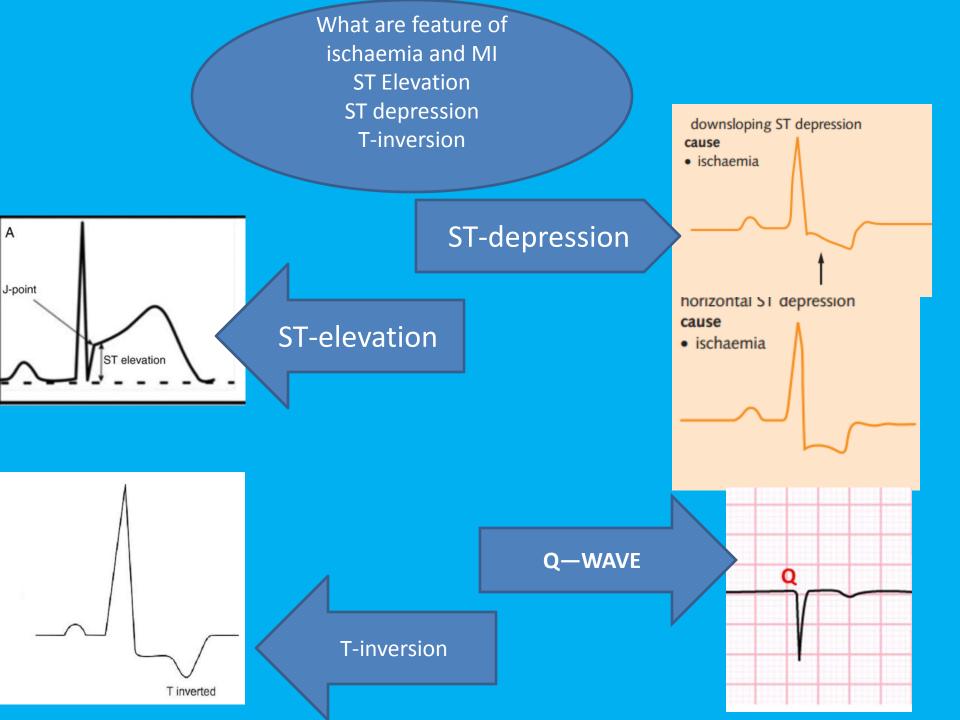




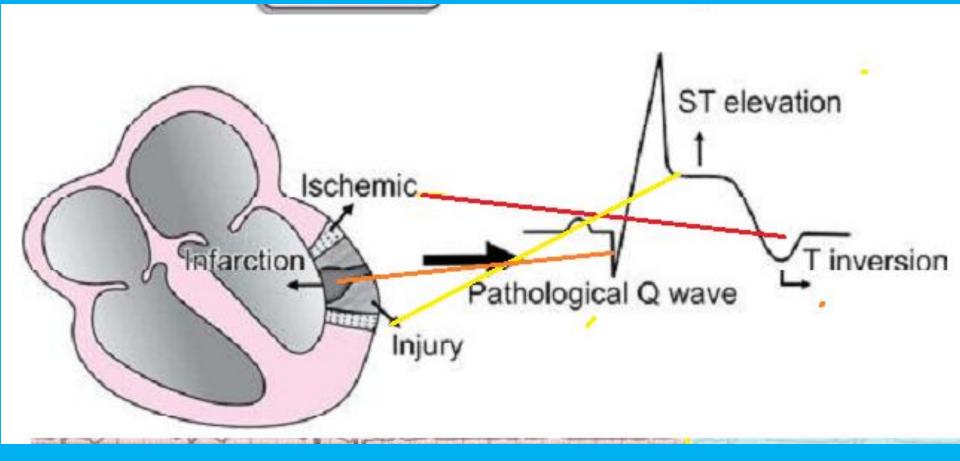
Concepts Behind Reciprocal Changes in Acute MI

It occurs when two electrodes look at the same acute MI from opposite angles, for example:

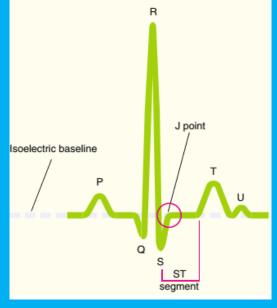
WHAT ARE THE CHANGES OCCURE IN ECG IN ISCHAEMIA & MI



Flow Chart 7.1: Pathology of acute myocardial infarction Coronary vessel block Diminished oxygen supply Ischemia Myocardial cells use up its oxygen reserves Initiates anaerobic metabolism Injury Causes acidosis Increased harmful metabolites in the cell Infarction Cell death Infarction Coronary vessel block Angina Coronary artery spasm Infarction Transient ST segment depression ST segment elevation



ST –**Segment change**



How ST-segment	The ST segment is measured from the end of the S wave to the start
measure	of the T wave
What is "J"	The J point represents the junction where the S wave meets the
	isoelectric baseline
What is it indicate	The ST segment represents the gap between ventricular
	depolarisation and repolarisation
Why ST segment	The ST segment is at the baseline because only an even exchange
normally remain in	of charges is taking place. That means During this period no
base line	additional electrical signals can pass through the myocardium

Why ST elevation

Ions normally move only through specialized ion channels in the myocardium. The ion channels require energy to function, and in ischemic injury loss of energy production these channel not function Cell death from severe ischemia generates holes in the cells, and ions can flow freely in and out bypassing the channels completely. So there is increase current flow toward injured area. that why corresponding lead shows ST elevation

In addition to ischemia, inflammation, drugs, electrolyte abnormalities, and genetic variation can each affect the normal function of these cardiac channels. ST elevation can also be seen in bundle branch block, hypertrophy, and pericarditis

Injury

As the zone of injury does not depolarize completely it remains more positive than the sourrounding tissue leading to ST segment elevation.

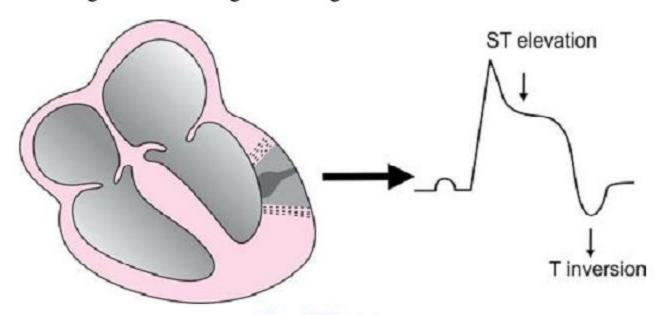
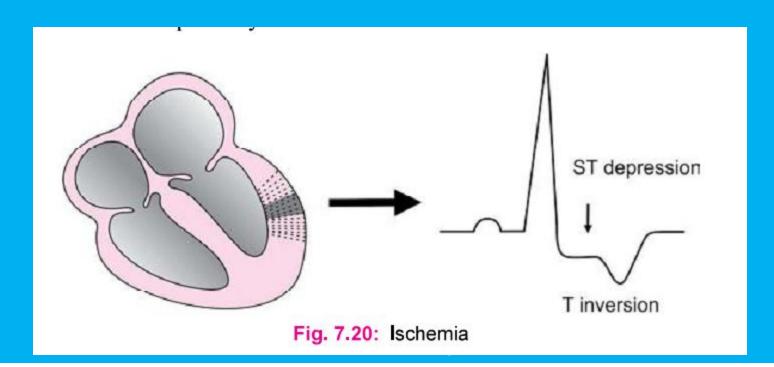


Fig. 7.21: Injury

T-wave and T –inversion

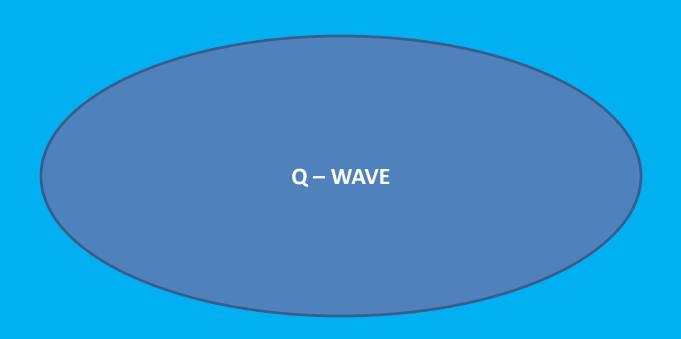
T wave indicate	repolarization of the ventricle
T wave morphology	the T wave is positive In most leads
	Negative in lead aVR
	Lead V1 may have a positive, negative, or
	biphasic (positive followed by negative, or vice versa)
	Isolated negative T wave in lead III, aVL, or aVF.
Why T is upright	Repolarization is negative current
	Repolarization of the ventricle happens in the opposite direction
	of depolarization that repolarization from apex to base (opposite
	to the direction of depolarization) but also a repolarization from
	epicardium to endocardium (opposite to the depolarization from
	endocardium to epicardium)
	This double reversal in repolarization directions account for the
	upward T wave, that is a T-wave in the same direction as the QRS
	complex

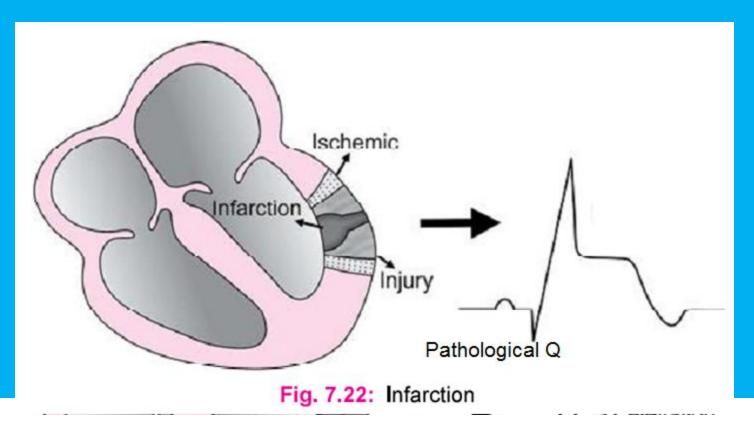


Ischemia

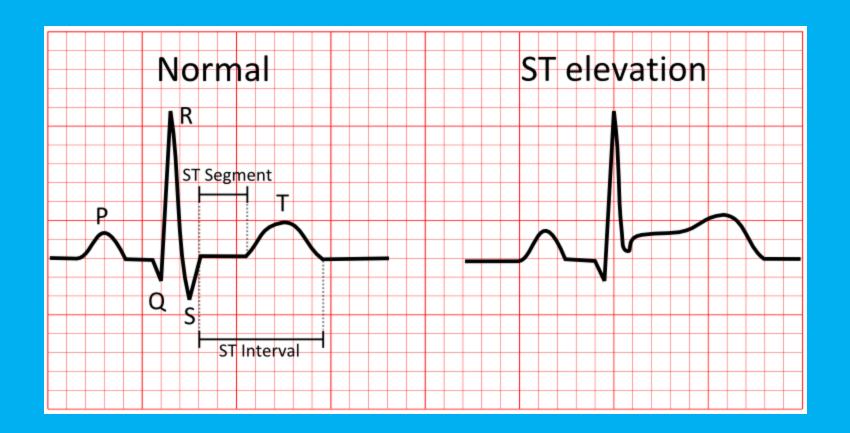
It affects a wedge shaped section of the heart, the apex facing the epicardium and the base facing the endocardium.

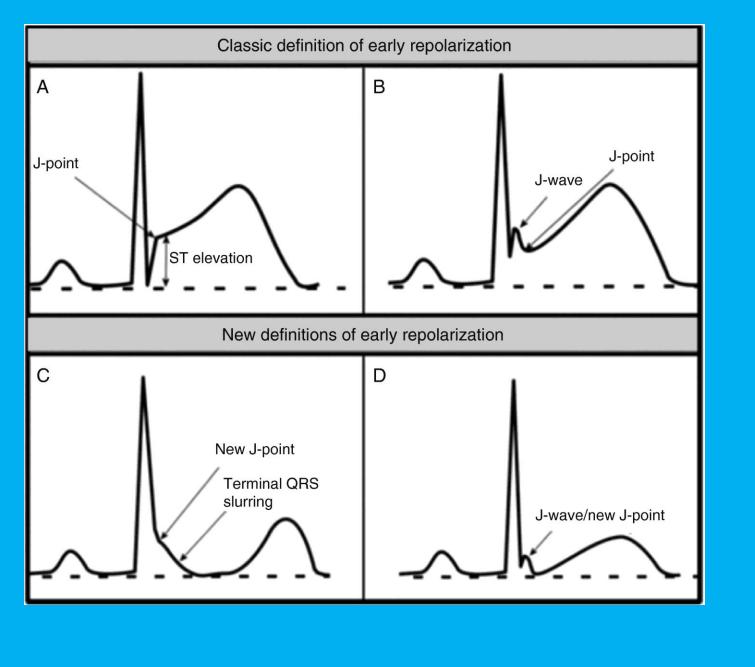
This area is more negative than the surrounding normal tissue leading to ST depression. Inverted T waves are seen in ischemia because repolarization occurs along the abnormal pathway.

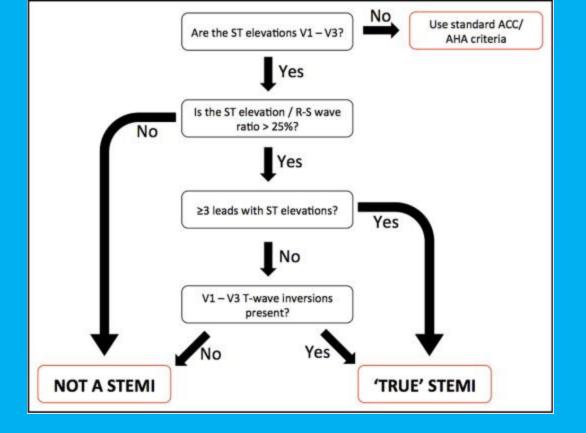


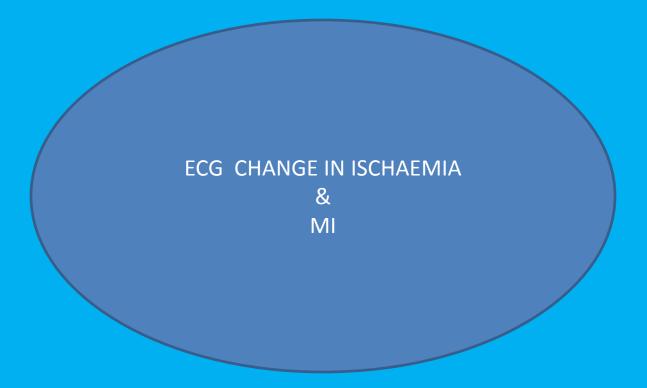


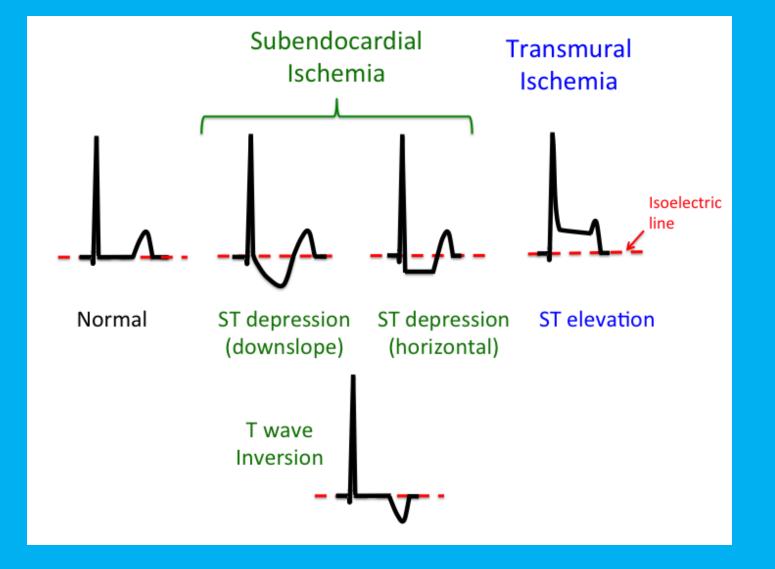
Indicates dead tissue. As the tissue is dead they do not generate any action potential and so it is electrically neutral. This electrically neutral area acts like a window in the myocardial wall. Through which an electrode can see the opposite wall. The positive vector of the other wall heading away from the electrode produces the pathological Q wave. The formation of the rest of the complex results from the surrounding zone of infarcts and injury.

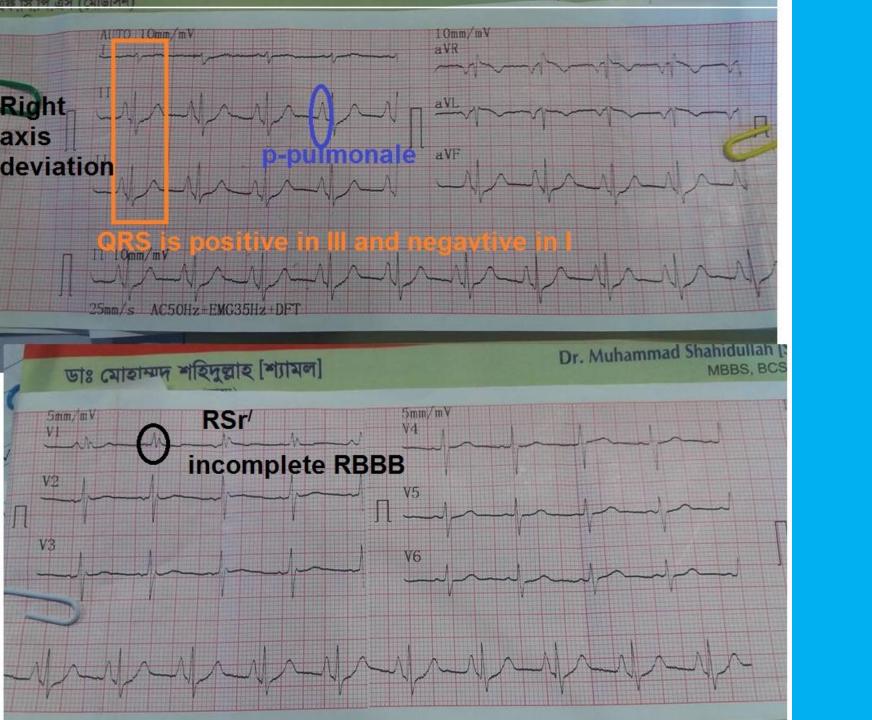


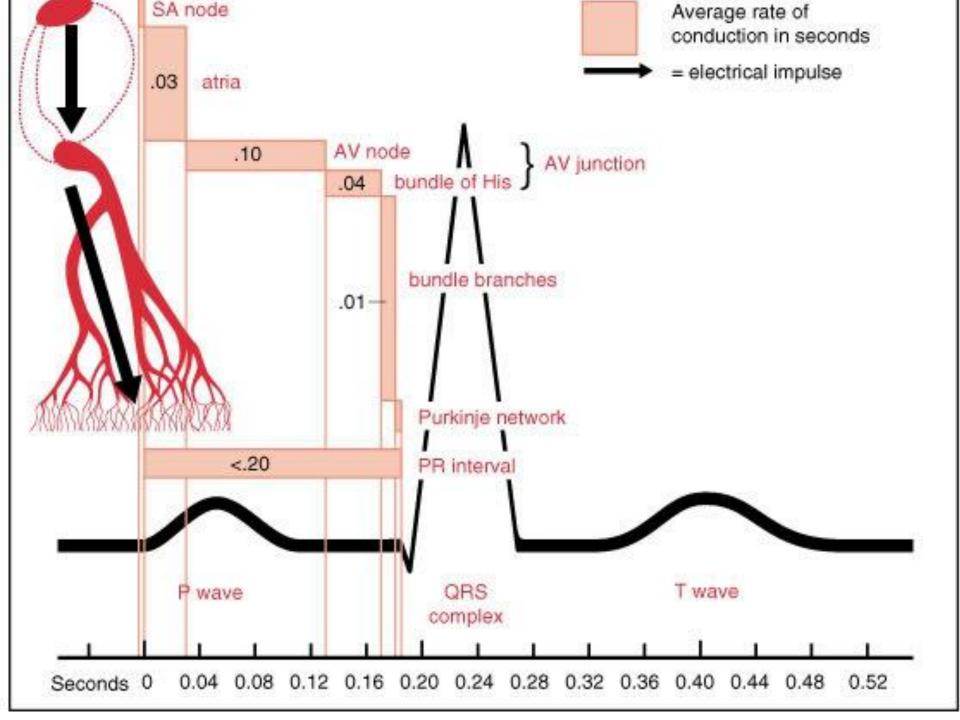












Blood supply to the heart:

- Right coronary artery inferior aspect of heart and right ventricle
- Left anterior descending Septal and anterior heart
- Left circumflex Lateral wall of left ventricle

It is important to keep these in mind when interpreting an ECG. Different leads focus on different aspects of the heart, and ischaemic changes in these leads indicate the location of the affected area:

- Anterior I and aVR
- Anterior-septal V1 and V2
- Anterior-lateral V3-6
- Inferior II, III and aVF

There are 3 phases of infarction:

- Acute phase characterised by ST segment elevation and tall T waves in affected area (hyperacute T waves may even precede ST elevation). NB in full thickness infarcts there may be reciprocal ST depression and T wave inversion opposite leads.
- Evolving phase (hours to days after) ST segment starts to return towards baseline, accompanied by inversion of T waves.
- Resolving phase (weeks to months after) partial or complete recovery from ST and T changes, may have persisting q waves if infarction. (If ST segment elevation persists, this may be indicative of aneurysm formation).

NB ST elevation in all leads is seen in pericarditis

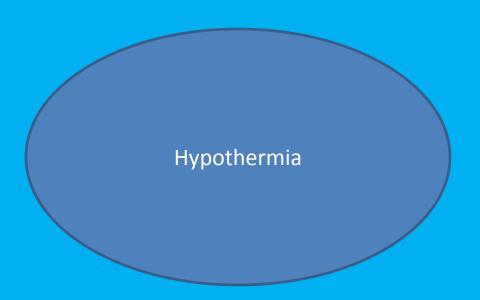
ST elevation is relevant if >1mm in limb leads and >2mm in chest leads.

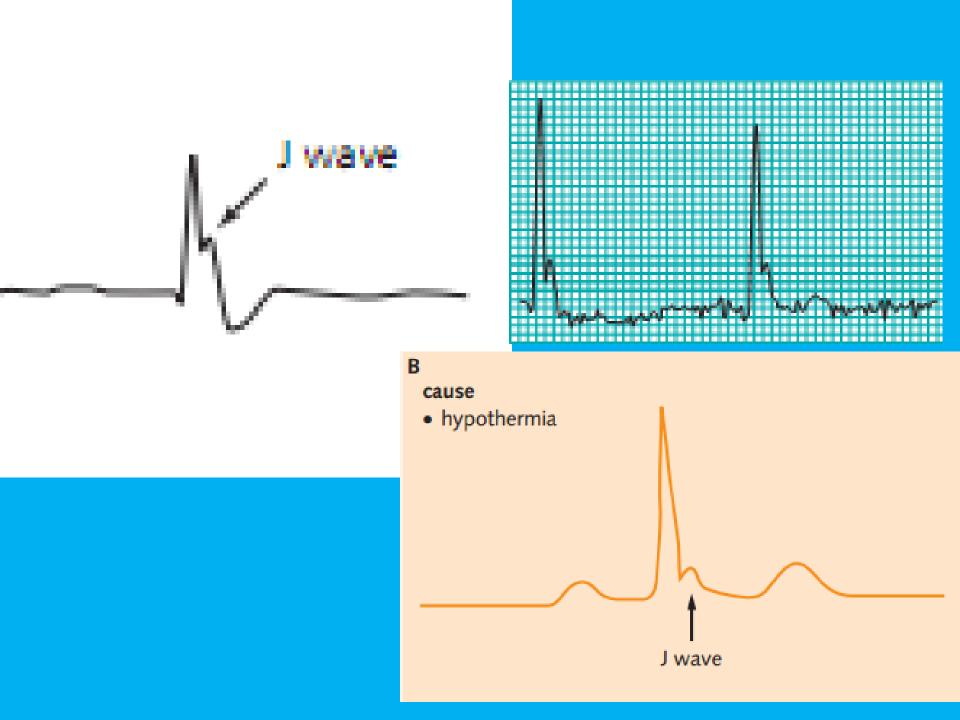
If there is no infarct, transient ischaemia is shown by ST segment depression

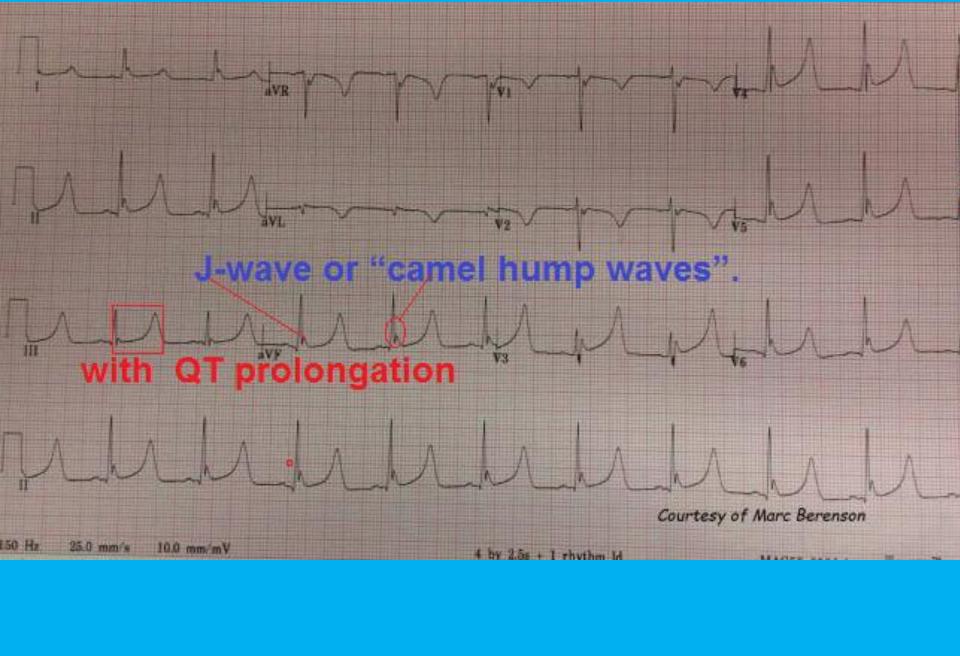
Blood supply to the heart:

- Right coronary artery inferior aspect of heart and right ventricle
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- Left circumflex Lateral wall of left ventricle
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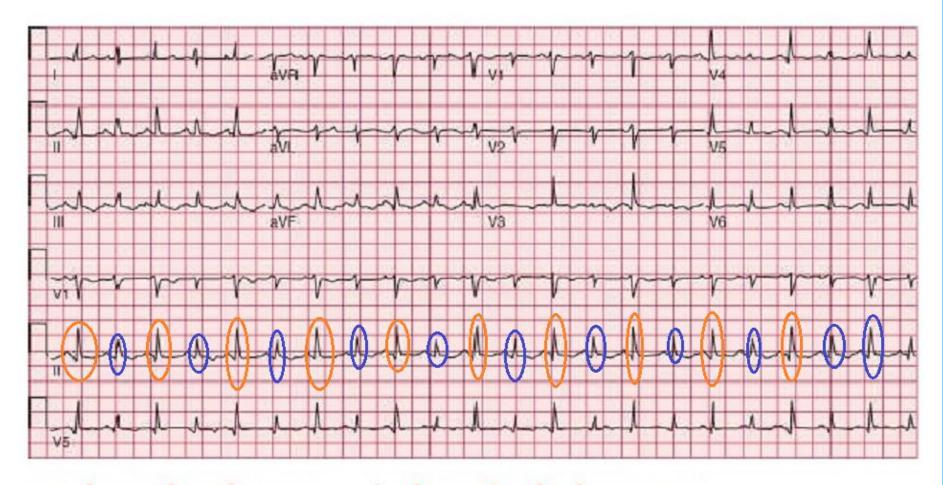
NB ST elevation in **all** leads is seen in pericarditis
ST elevation is relevant if >1mm in limb leads and >2mm in chest leads.
If there is no infarct, **transient ischaemia** is shown by ST segment **depression**







Pericardial effusion

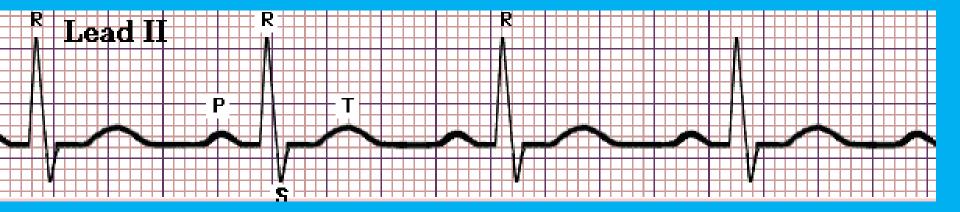


reduced voltage and electrical alternans



SVT
VT
WPW
AF
Atrail flutter
AV—block
Sinus tachy and brady arhythmia

Sinus Rhythm



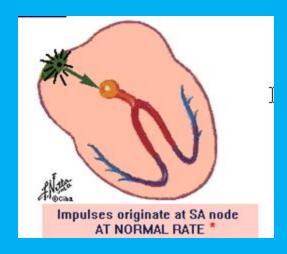
Rate: Normal (60–100 bpm)

Rhythm: Regular

P Waves: Normal (upright and uniform)

PR Interval: Normal (0.12–0.20 sec)

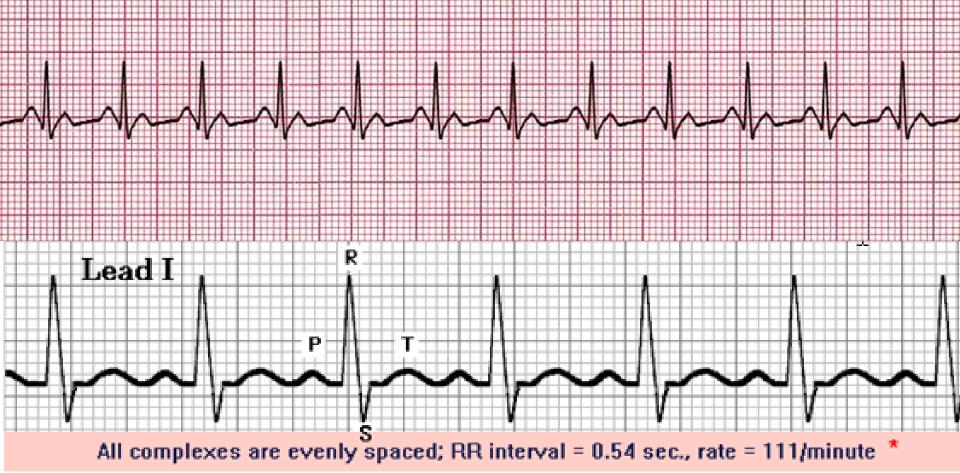
QRS: Normal (0.06–0.10 sec)



Add a ECG of sinus rhythma

Sinus tachycardia

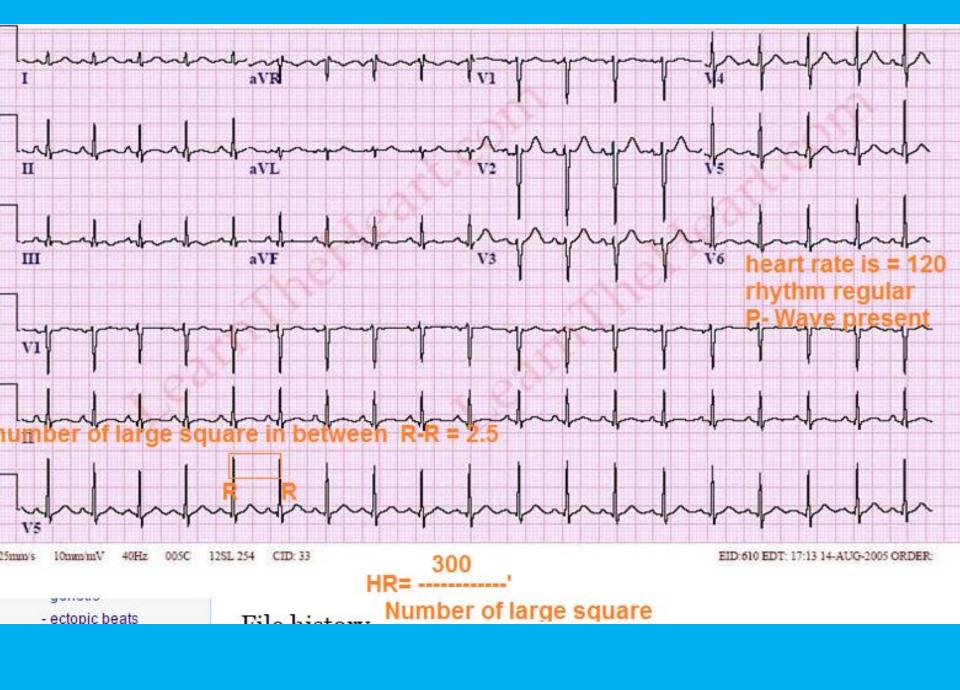
SINUS TACHYCARDIA



Rate: fast (>100 bpm)

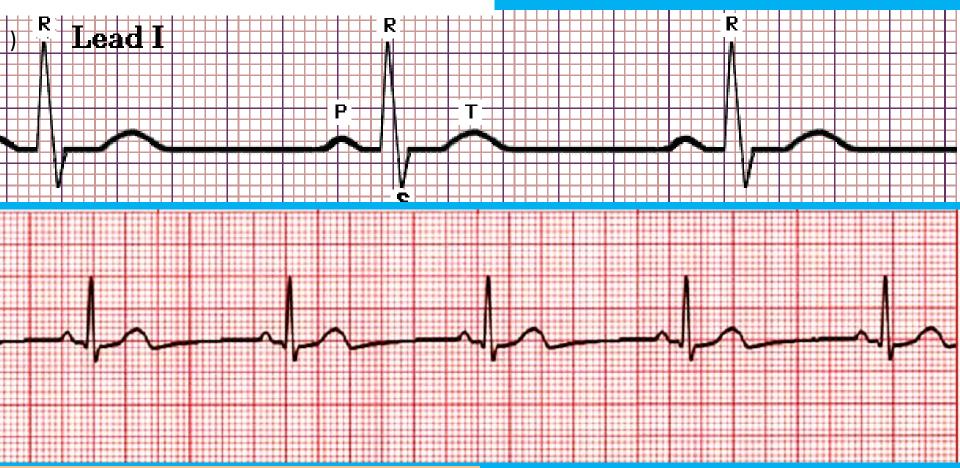
Rhythm: Regular

P Waves: Normal (upright and uniform) PR Interval: Normal (0.12–0.20 sec)



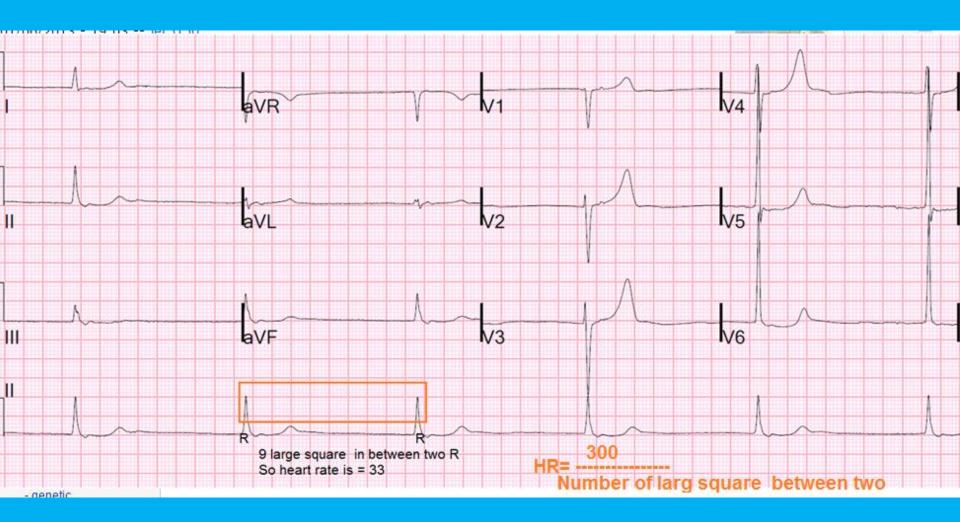
Causes of sinus tachycardia

SINUS BRADYCARDIA

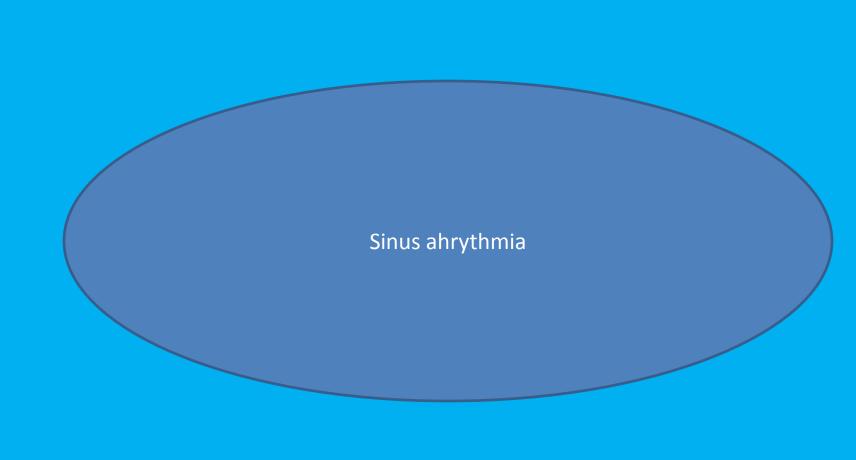


Rate: Slow (60 bpm) Rhythm: Regular

P Waves: Normal (upright and uniform) PR Interval: Normal (0.12–0.20 sec)



Causes of sinus bradycardia



SINUS ARRYTHMIA

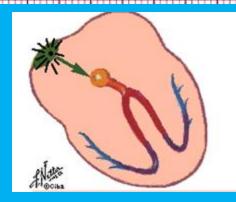


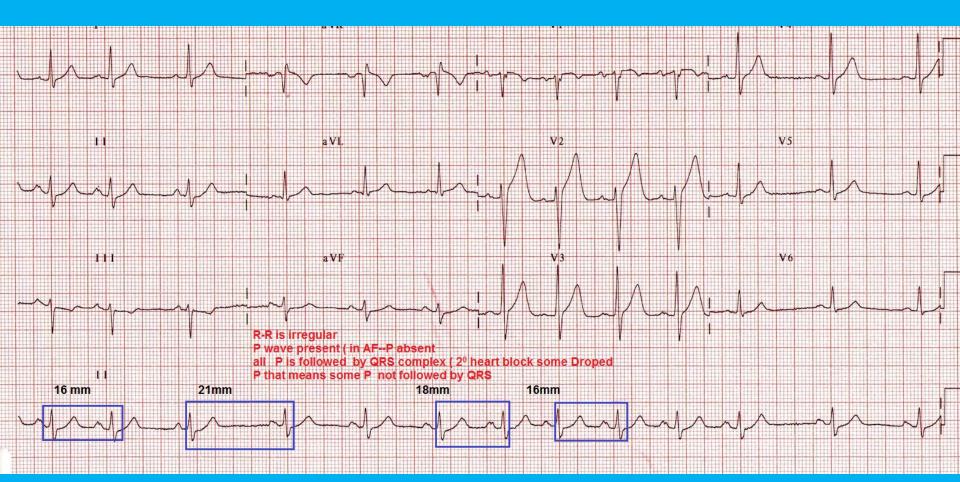
PR Interval: Normal (0.12–0.20 sec) QRS: Normal (0.06–0.10 sec)

The SA node discharges irregularly

Quick clue:

- □R-R interval is irregular,
- □PR Interval fixed and P=QRS (every is P is followed by QRS complex)



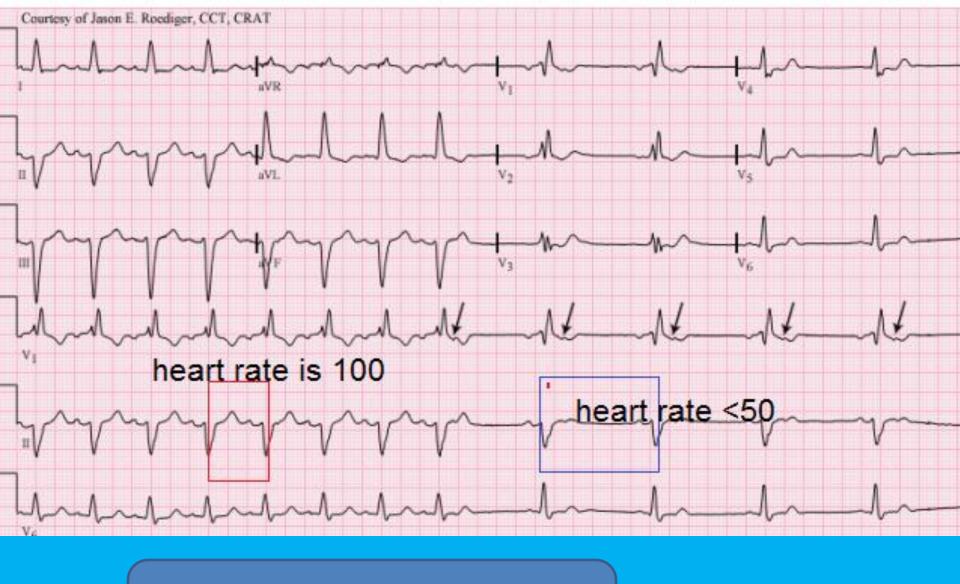


□R-R interval is irregular, □PR Interval fixed and P=QRS (every is P is followed by QRS complex) Sick sinus syndrome

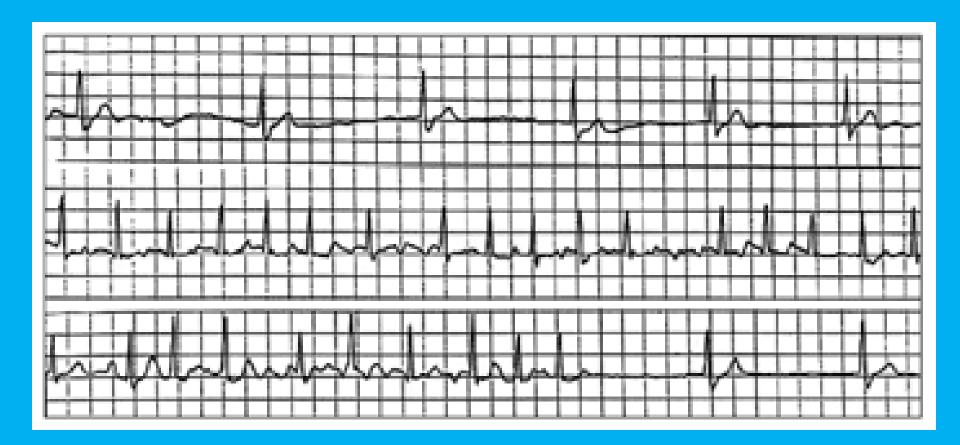
In sick sinus syndrome there is a malfunctioning sinus node. Several arrhythmias can result from this:

- Symptomatic slow sinus bradyardia in the absence of medication
- ➤ Sinus Arrest or exit block
- Combinations of sinoatrial and atrioventricular conductions disturbances
- ➤ Brady-tachycardia syndrome; typically there is sinus bradycardia, sinus arrest or SA-block which is alternated by periods with fast or (ir)regular atrial arrhythmias (atrial fibrillation, atrial flutter, atrial tachyardia or sinus tachycardia).

Usually a pacemaker is implanted to prevent bradycardia and tachycardias are 'topped off' with anti-arrhythmics, such as beta blockers.



Tachy –brady syndrome





Heart Block

SA NODE BLOCK

- 1. Sinus pause
- 2. Sinus arrest

Sinus arrest with atrial escape beat Sinus arrest with nodal or junctional escape beat Sinus arrest with ventricular escape beat

AV NODE BLOCK:

1ST degree

2nd degree

3rd degree of complete heart block

Bundle branch block:

RBBB

LBBB

SA NODE dysfunction

- 1. Sinus pause
- 2. Sinus arrest

Sinus arrest with atrial escape beat Sinus arrest with nodal or junctional escape beat Sinus arrest with ventricular escape beat

- 3.sino-atrial block
- 4. Sick sinus syndrome

The SA node consists of	A central core of pacemaking cells ("P	dysfunction of This cell leads to	
two main groups of cells:	cells") produce the sinus impulses	sinus pauses and sinus arrest.	
	transitional cells ("T cells") that transmit	Failure of the T cells to transmit	
	the sinus impulses out into the right	the impulse. This leads to sino-	
	atrium	atrial exit block.	
Sinus pause	A sinus pause is defined as the transient absence of sinus P waves on the ECG that may last from two seconds to several minute		
Sinus arrest	A sinus arrest is defined as absence of normal p-waves on the		
	ECG for more than 2 seconds		
	Or if sinus pause persist more than 2 second is called sinus		
	arrest		
	After a brief period either the sinoatrial node resumes normal pacing, or another pacemaker begins pacing. If a pacemaker other than the sinoatrial node is pacing the heart, this condition is known as		
	an <u>escape rhythm</u>		
	If no other pacemaker begins pacing during an episode of sinus		
	arrest it becomes a <u>cardiac arres</u> t		
Three escape rhythm	it is in the following order:		
	1. Atrial escape (rate 60–80): normal P morphology is lost		
	2. Junctional escape (rate 40–60): P is inverted or retrograde		
	3. Ventricular escape (rate 20-40): no	P wave, wide, abnormal QRS.	

an SA block	The electrical impulse is delayed or blocked on the way to the atria, thus delaying the			
	atrial beat.			
	The sino-atrial node continues to depolarise normally. However, some of the sinus			
	impulses are "blocked" before they can leave the SA node, leading to intermittent failure			
	of atrial depolarisation (dropped P waves).			
	First Degree SA block	Delay between impulse generation and transmission to the atrium		
		This abnormality is not detectable on the surface ECG		
	Second Degree SA	The P-P interval progressively shortens prior to the dropped P		
() 	block, Type I	wave. This is also known as type I sinus exit block		
	(Wenckebach)	gradual reduction in the P to P interval (the distance		
		between one P wave and the next). This results in a pause, the cycle		
		then continues		
	Second degree SA	there is no shortening of the P to P interval but instead an		
	block Type II	unexpected absence of a P wave and subsequent QRS complex The		
		pause is multiple of the P to P interval		
	A third degree	Looks very similar to a sinus arrest.		
	sinoatrial block	However, a sinus arrest is caused by a failure to form impulses. A		
		third degree block is caused by failure to conduct them.		
		It is followed by a long pause that is not a multiple of the P-R		
		interval.		
		The pause ends with a P wave, instead of a junctional escape		

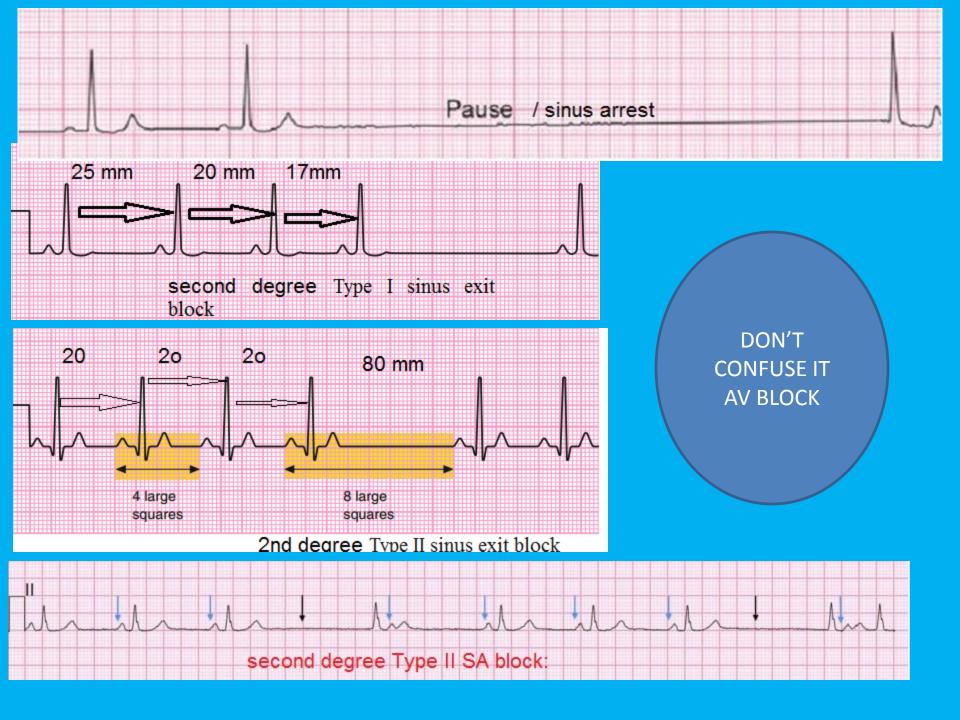




Fig. 8.13: Sinus arrest with atrial escape beat



Fig. 8.14: Nodal or junctional escape beat

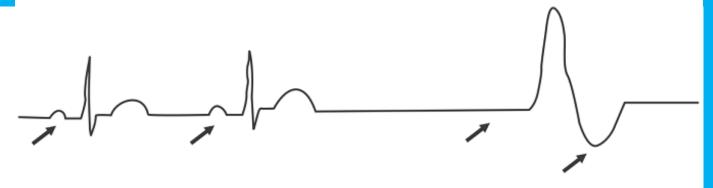


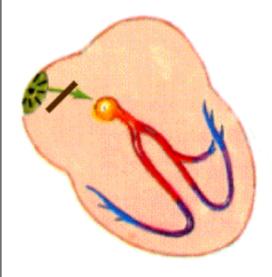
Fig. 8.15: Ventricular escape beat

Sinus arrest

Sinus arrest with atrial escape beat
Sinus arrest with nodal or junctional escape beat
Sinus arrest with ventricular escape beat

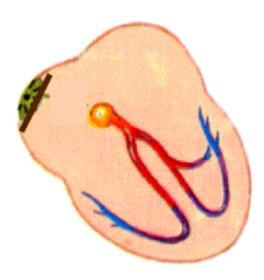
What is happening in a Sinus Block and Sinus Arrest?

SA Block



Pause is multiple of R-R Intervals

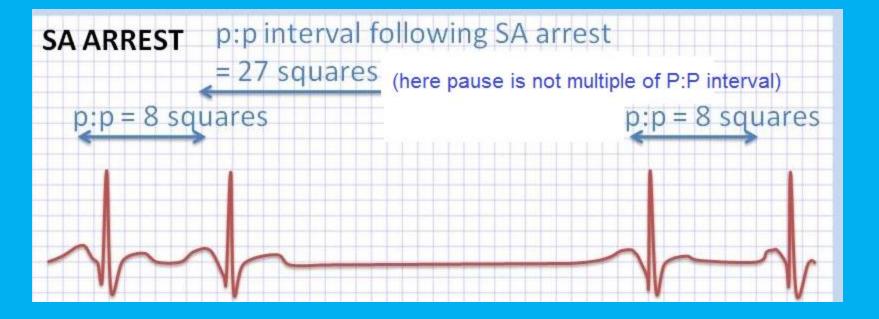
Sinus Arrest



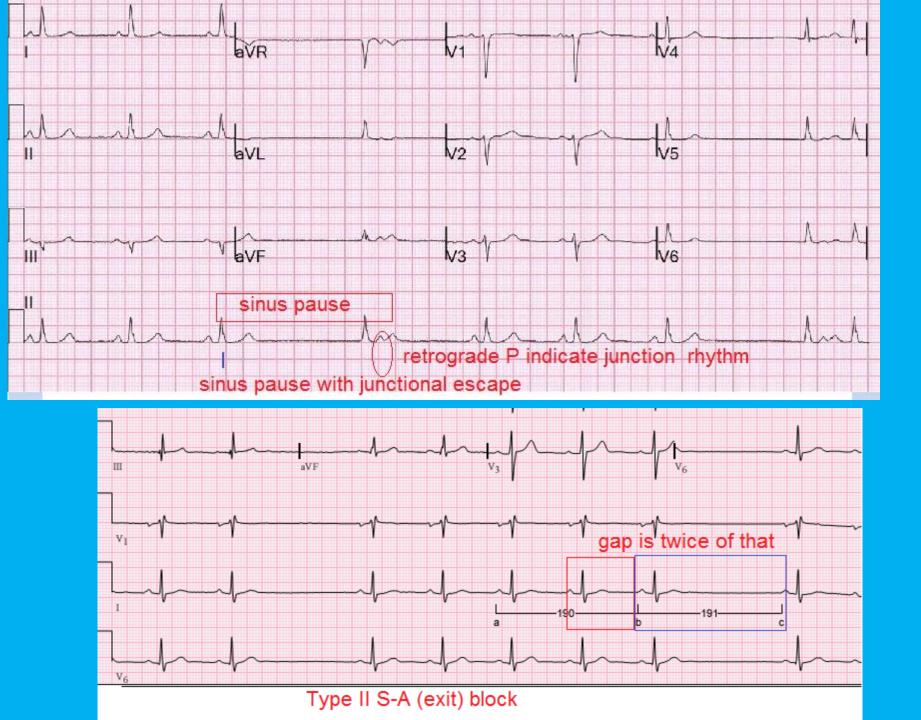
Pause NOT multiple of R-R Intervals

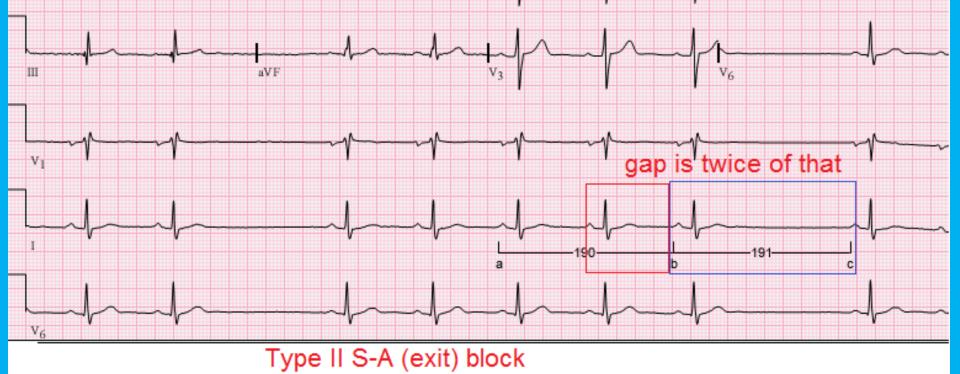
NOTE: With SA Block the R-R interval measurement can be within plus or minus 2 small boxes. If it is greater than the plus or minus 2 small boxes it is a sinus arrest.

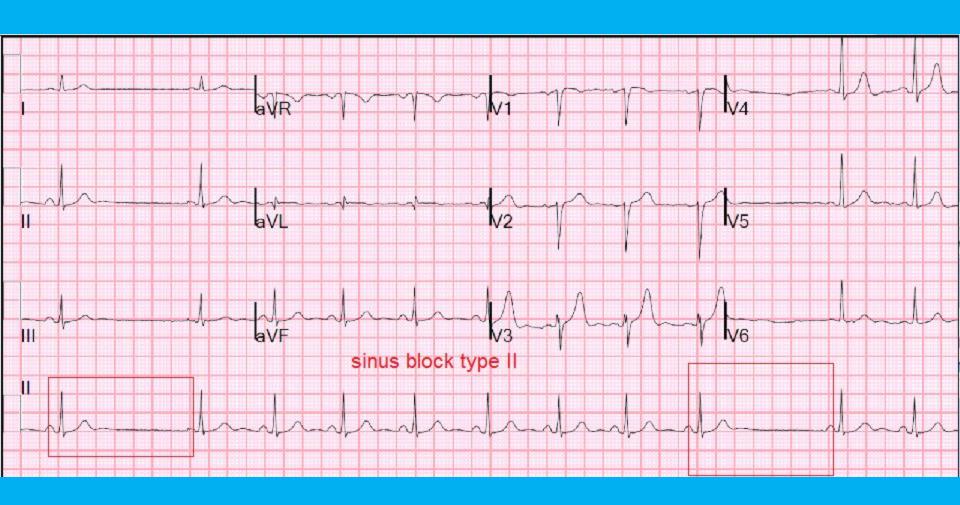
p:p interval following SA block = 16 (8x2=16) here pause is multiple of P:P interval p:p = 8 squares p:p = 8 squares



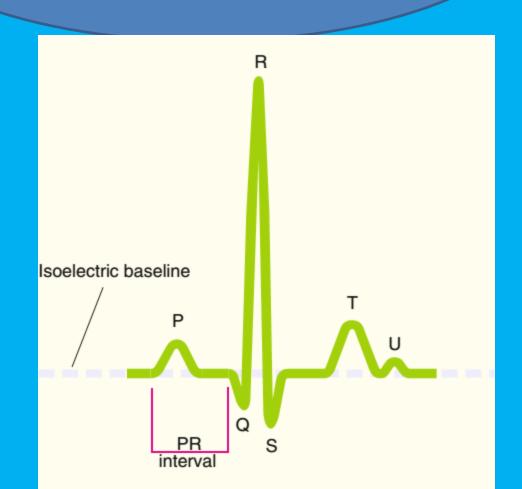








AV-NODAL BLOCK



PR interval

- •It is the distance between onsets of P wave to the beginning of Q wave
- •It is the time require for impulse to travel SA node to ventricle muscle
- Normal PR interval: 0.12 to .20

PR may be

```
Normal = (0.12 \text{ to } .20 \text{ sec or } 3 \text{ to } 5 \text{ mm})

Prolong = (> 5 \text{ mm}) 1^{\circ} heart block

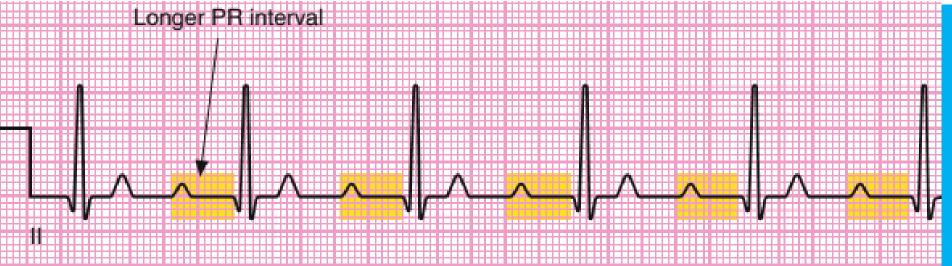
Short = (< 3 \text{ mm}) WPW syndrome

Variable = 2^{\circ}/3^{\circ} heart block
```

First degree Heart Block

1º heart block when PR interval is more Than 0.2 sec or > 5 mm

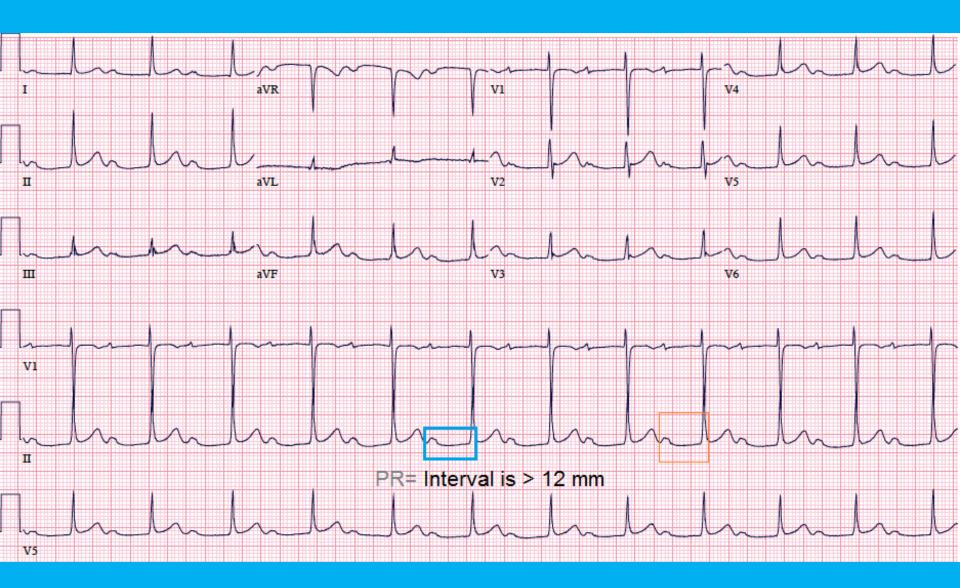




R-R = is regular
PR interval = is fixed and more than >0.2 sec or > 5mm
P = ORS complex, that means every P is followed by QRS

Cause of first degree heart block

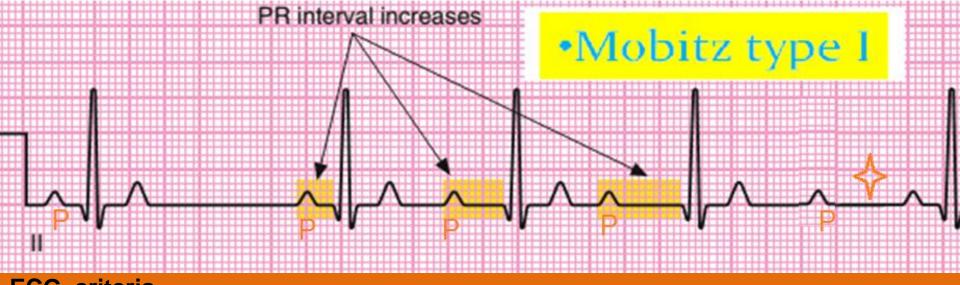
- □AMI /IHD
 □Acute rheumatic fever
 □Myocarditis
 □Hypokalaemia
 □Digitalis toxicity , quinidine ,B-blocker
 - \square No R_x
 - □R , of the primary cause



2nd degree Heart Block

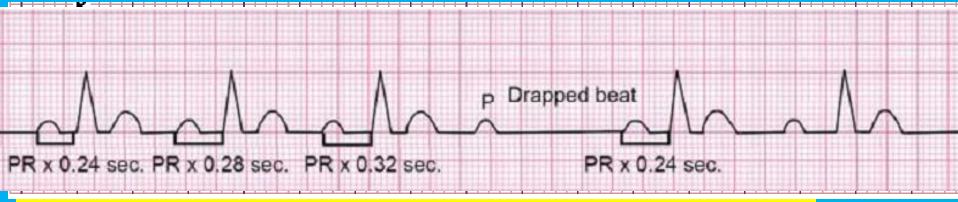
2nd degree heart block

Mobitz type I Mobitz type II



ECG criteria

Progressive lengthening of PR followed by absent of QRS complex. That meant one P is not followed by QRS complex and repetition of this cycle.



To remember □R—R = is irregular

 \square PR = is not fixed (progressive \uparrow PR)

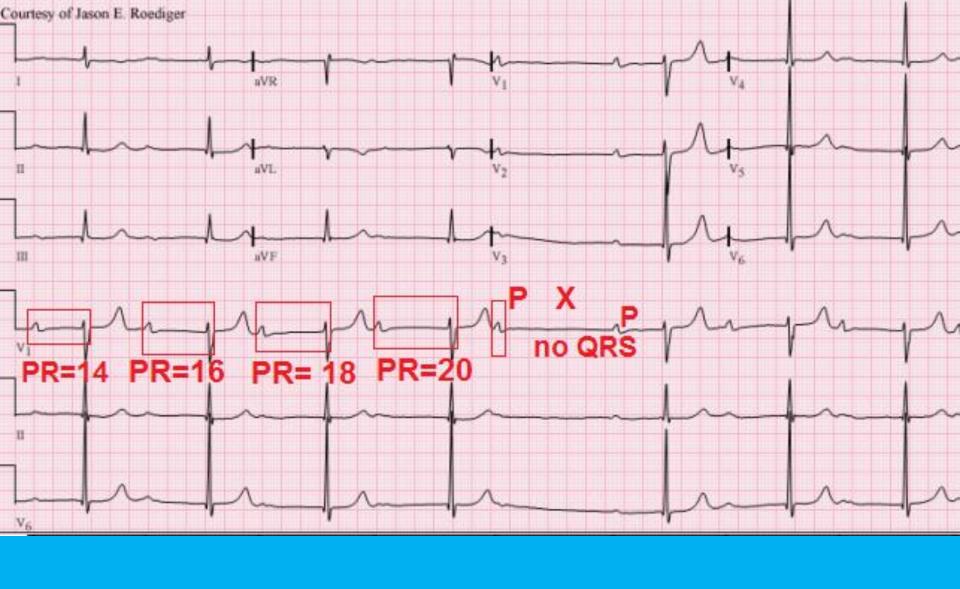
□P≠QRS), some p is not followed by QRS complex

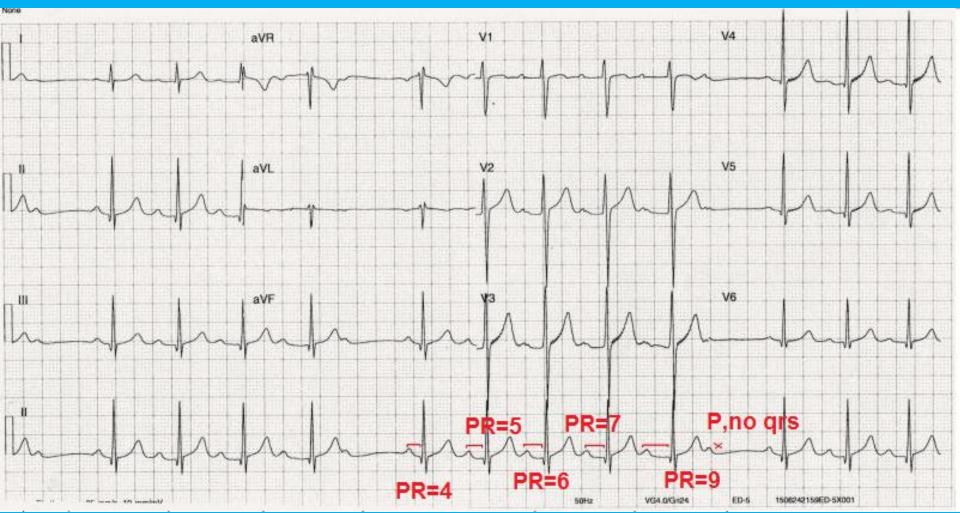
Cause:

- Physiological in athlete
- **❖Drug:** Digoxin
- Acute myocardial infarction (INF)

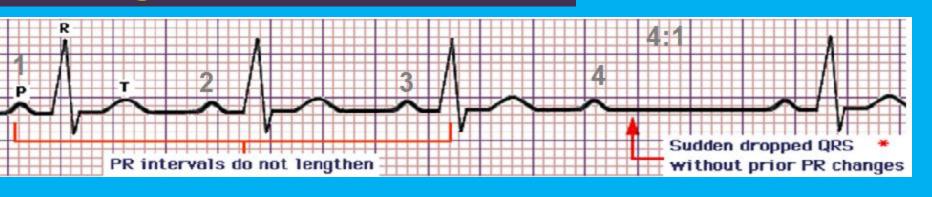
Clinical feature

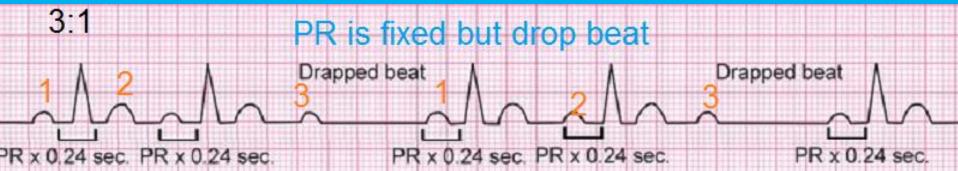
- ✓ Asymptomatic pulse is irregular (drop beat)
- ✓ Prognosis is good





2"degree hear block Mobitz type II



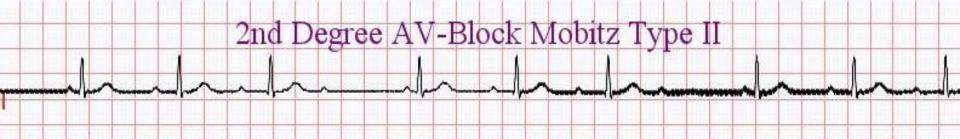


To remember

- >PR = is fixed (it is differentiate point from type I)
- R-R = is irregular (it is differentiate point from 1st & 3rd degree)
- > (P≠QRS) Some p is not followed by QRS complex,

It may follow 2: 1, 3:1 or haphazard (in 2: 1 every alternate P wave is conducted)

Site: HIS purking system



CAUSE: Acute MI

Associated inferior MI

if asymptomatic:

No R_x, close monitor

If symptomatic:

- Inj. Atropin 0.6 mg 1 vial IV stat @
- If fail uses temporary pacemaker
- The condition resolve with in 7 to 10 days

Prognosis:

- Complete heart block
- Stokes Adams syndrome
- Heart failure

If associated Anterior MI

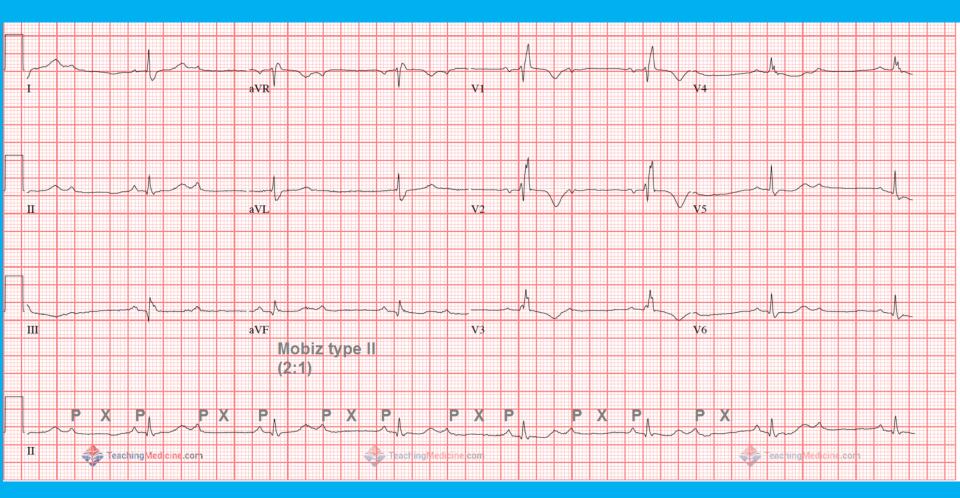
Temporary pacing followed by permanent pacing .(Because : chance of complete heart block)

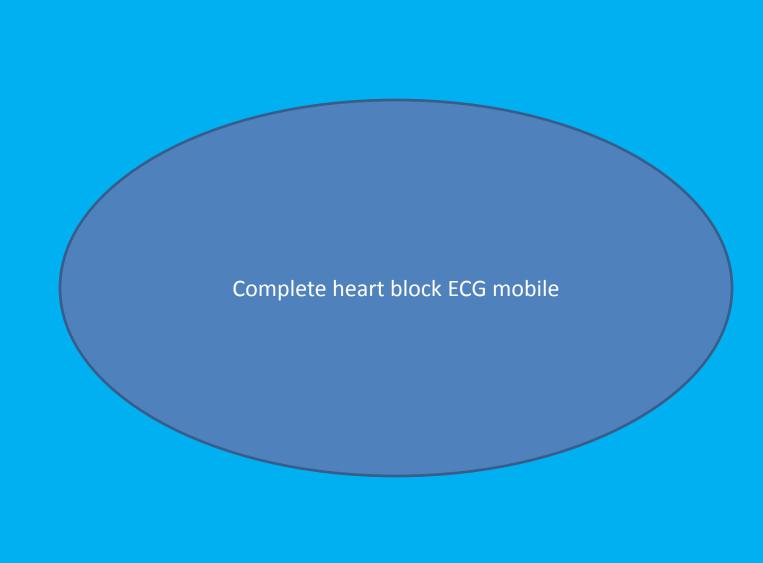
If patient presents with asystole, give

❖i. v. Atropine (3 mg) or

❖i.v. isoprenaline

oisoprenaline(2 mg in 500 ml 5% dextrose, infused at 10-60 ml/hour)may help to maintain the circulation until a temporary pacing electrode can be inserted.

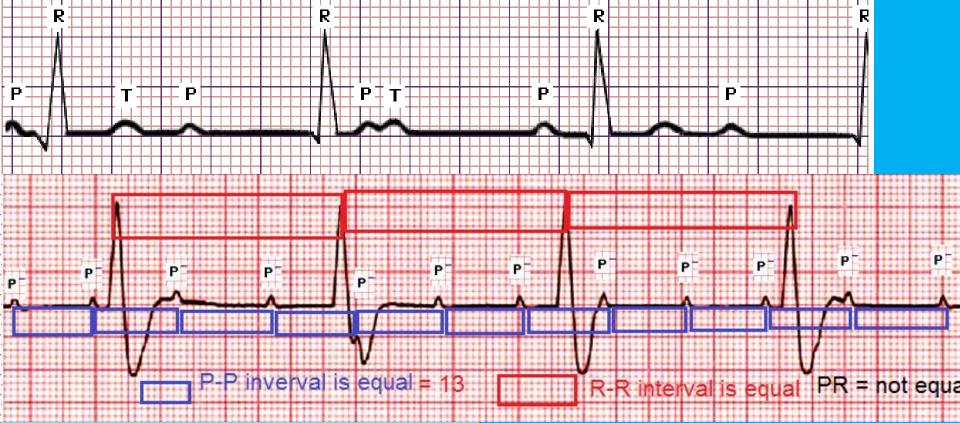




Complete or 3rd degree hear block

Clue to diagnosis

In brady-cardia ECG usually less than 40 where P—P AND R—R regular and there is no relation of P and QRS complex (u identify it by that PR is not fixed it is variable)



PR = is not fixed (variable)

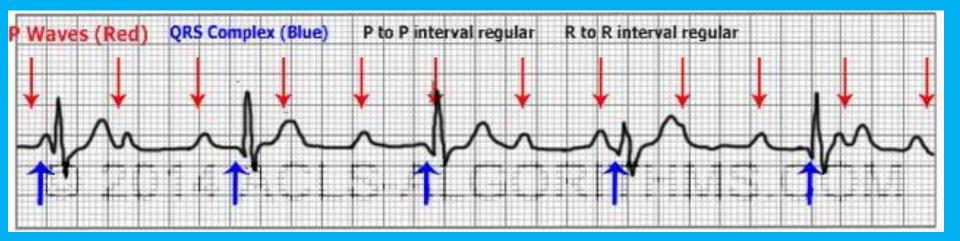
R-R = rregular

P—P =regular

P≠ QRS, no relation between P and QRS

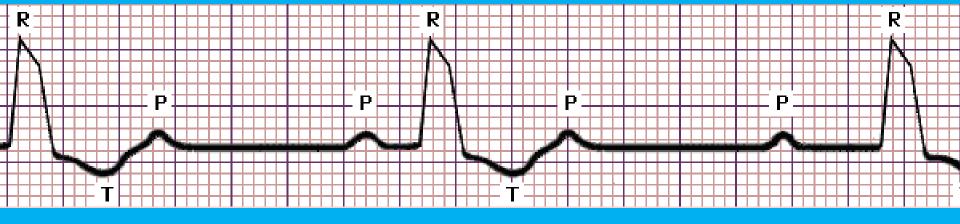
complex.

Brady-cardia



Most common cause

- ☐Myocardial infarction/ischaemia
- □Drugs (e.g. β-blocker, digoxin)
- □ Idiopathic fibrosis
- **□**Inflammation
 - **❖** Acute (e.g. aortic root abscess in infective endocarditis)
 - Chronic (e.g. sarcoidosis; Chagas disease,)
 - Trauma (e.g. cardiac surgery)
- **□**Congenital

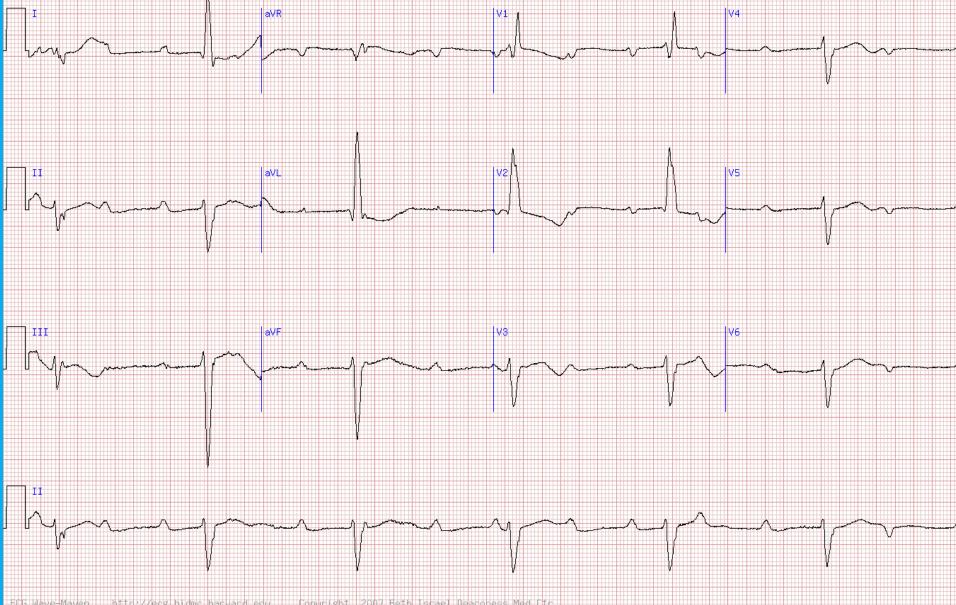


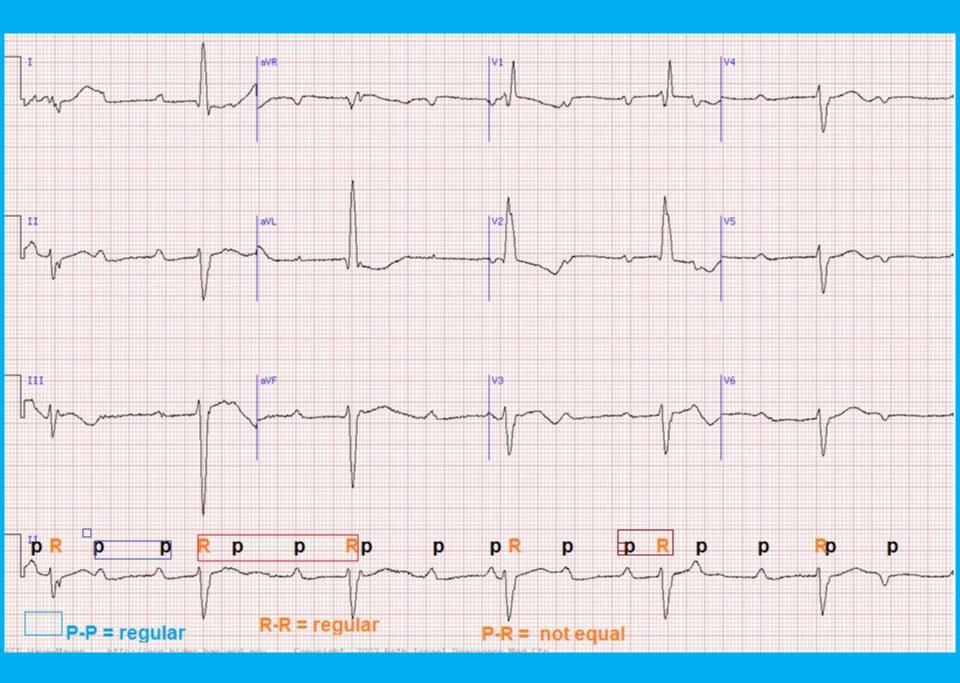
Clinical feature

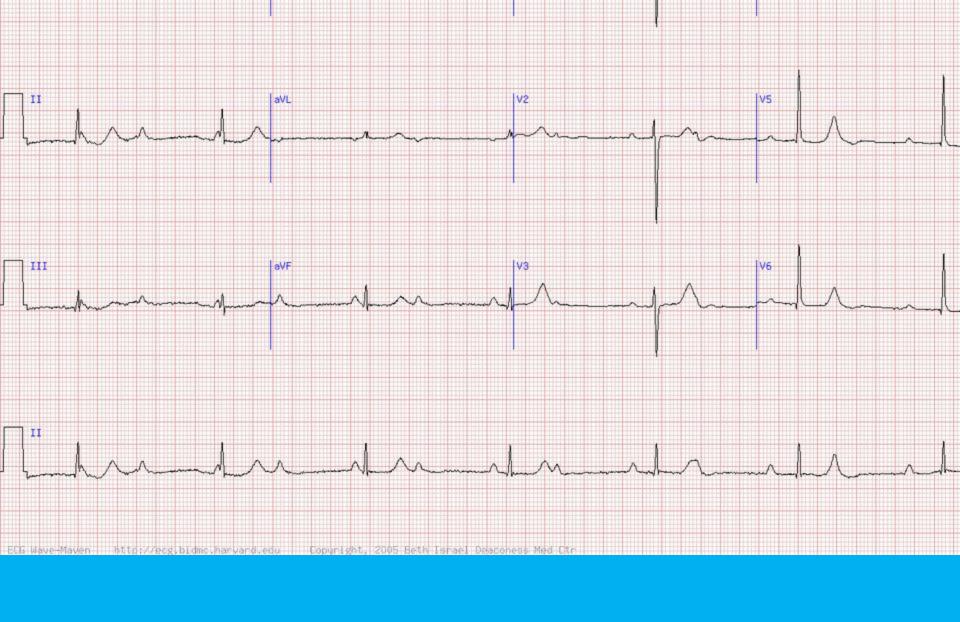
- □Dizziness . sudden loss of conscious , syncope , blackout
- □Pulse : Brady cardia 20 40 beat / minutes
- □BP : high SBP and normal Diastolic class
- □Neck vein : cannon wave

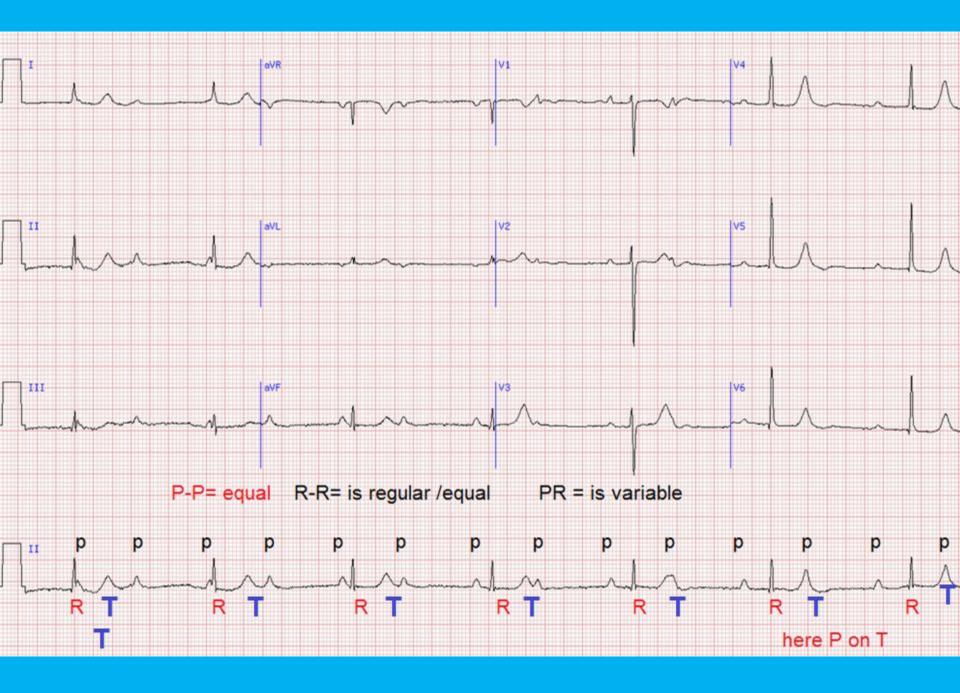
Treatment

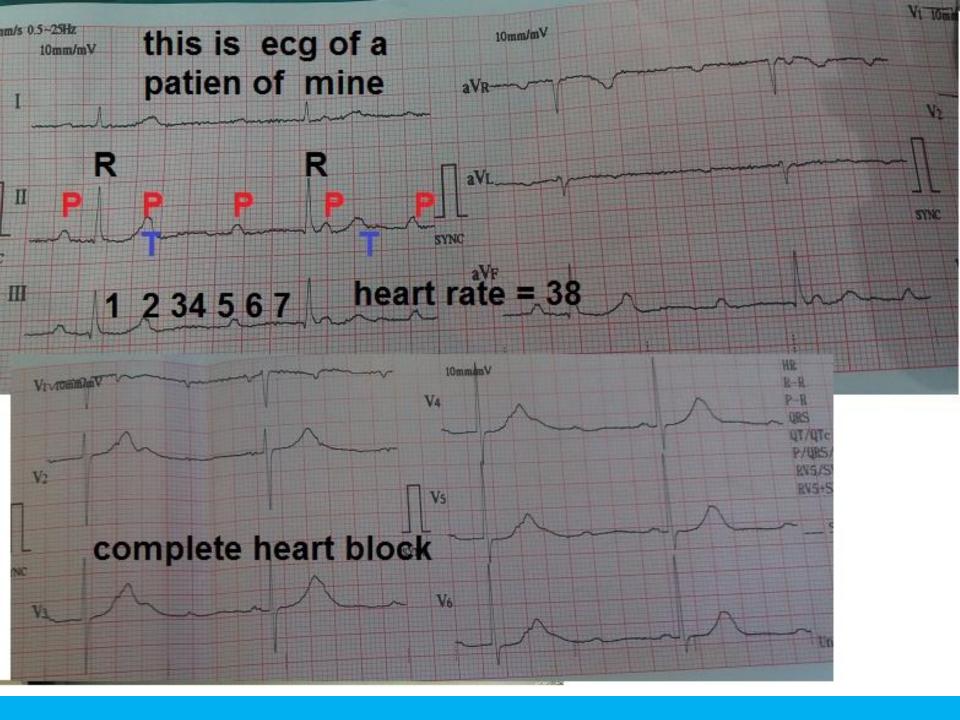
- **→** Symptomatic
- > Permanent pace maker
- >Treatment of cause

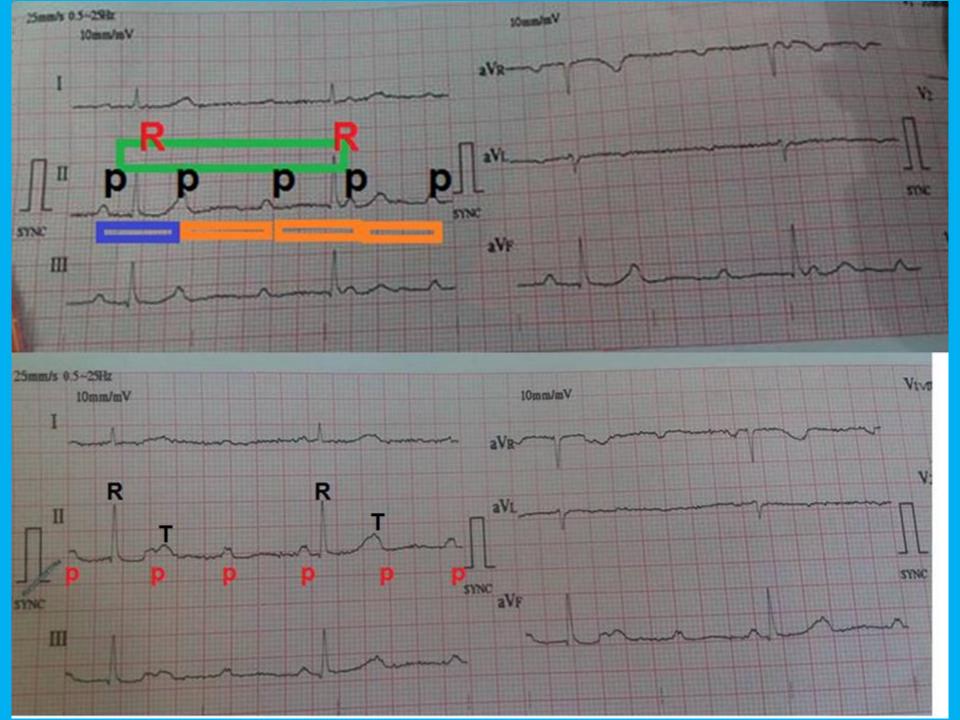


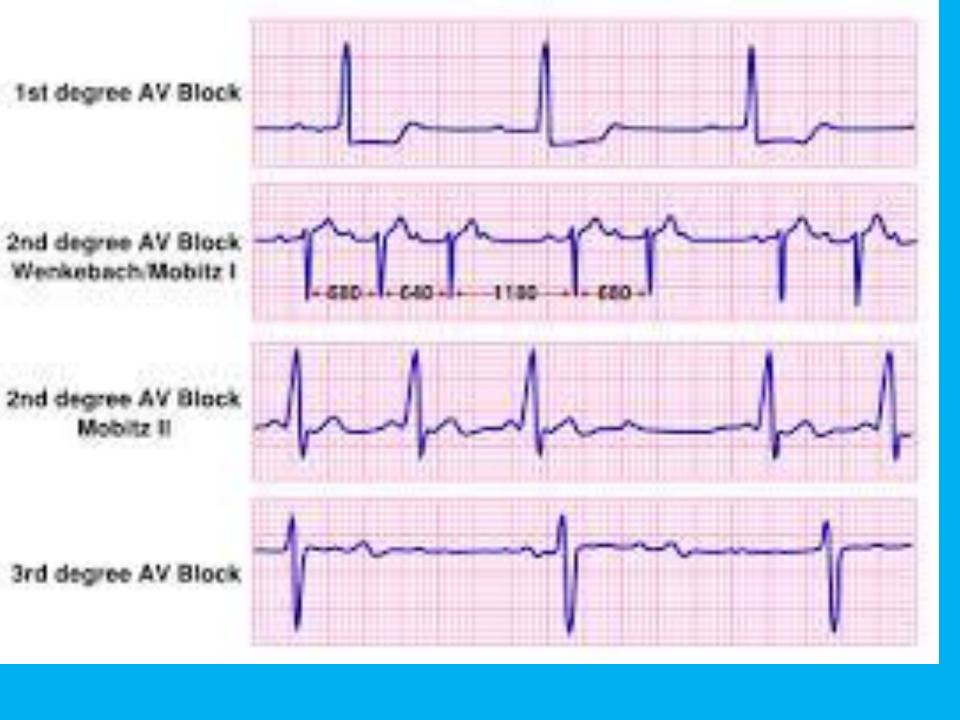












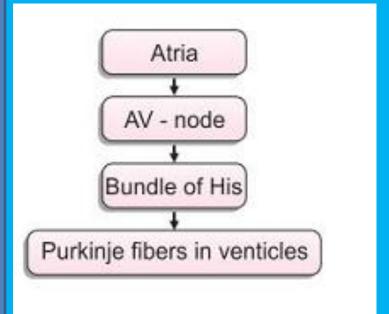
Premature beat

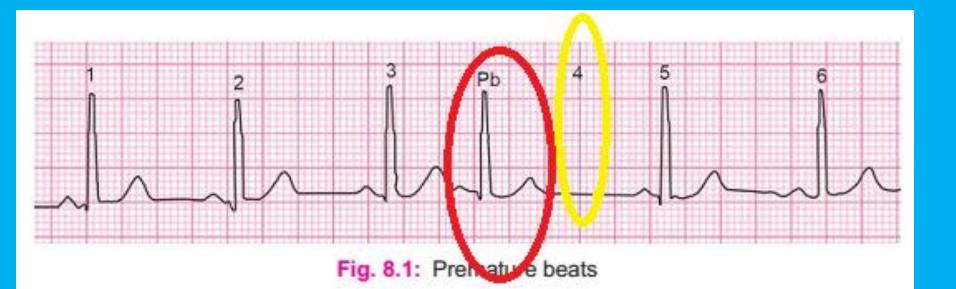
Premature Beats/Ectopic Beats/Extra -systole

It is the beat that is arising from an ectopic focus outside the SA node and occurring before the next sinus beat. It may arise from:

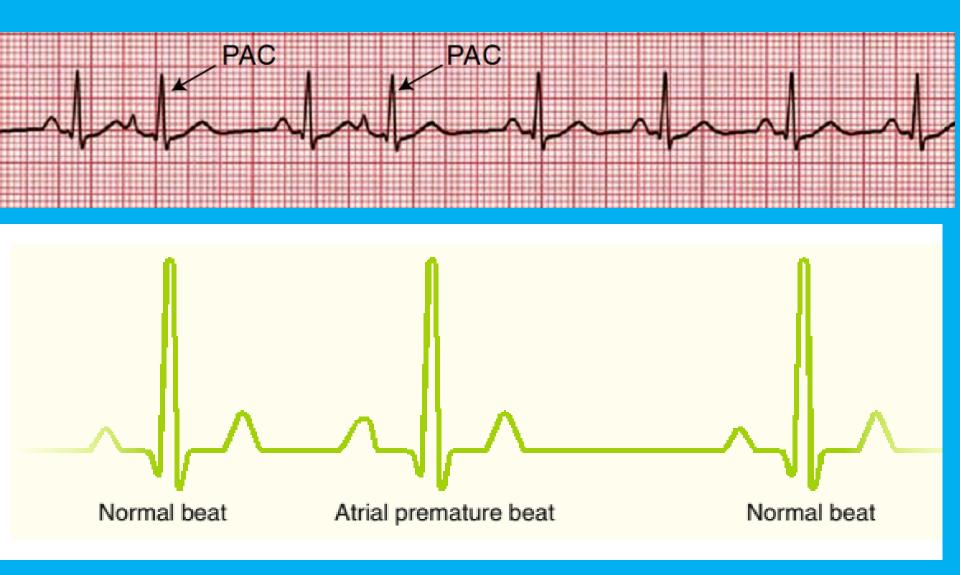
- I. Atria
- II. Nodal
- III. Ventricular

It can arise from either of the ones mentioned above because pace maker are located in the following order





pb=Premature beat this beat was schedule for lebel 4 but it comes earlier



Depending upon the site of origin of premature beat it is classifid as—

- I. Supraventricular premature beat / atrial or nodal
- II. Ventricular premature beat.

Compensatory pause

It is defied as the pause between the premature beat and the next sinus beat. Compensatory pause can be of two types they are:

- 1. Complete compensatory pause
- 2. Incomplete compensatory pause.

Complete compensatory pause: If the compensation occurs exactly for the missed beat and the third sinus beat occurs exactly where it would otherwise occur, then it is a complete compensatory pause.

Incomplete compensatory pause:

If the beat following the premature beat occurs before the next expected beat, then it is incomplete compensatory pause.

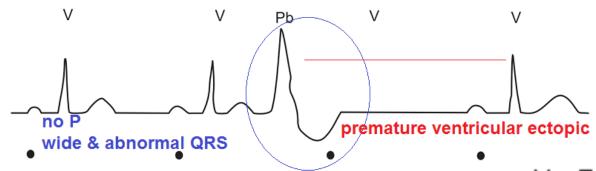
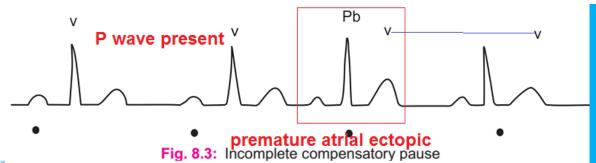
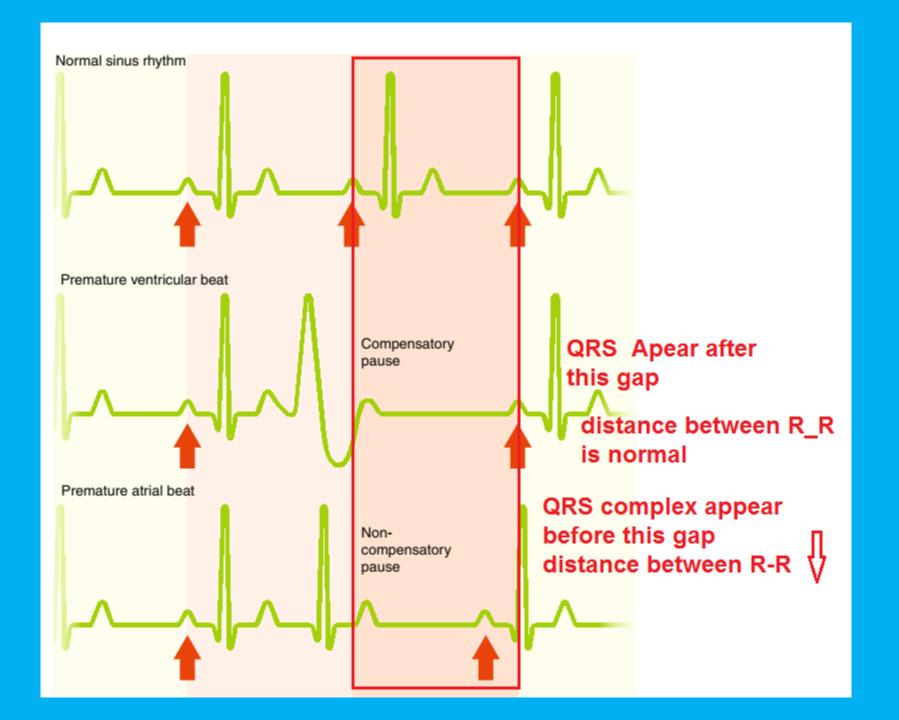


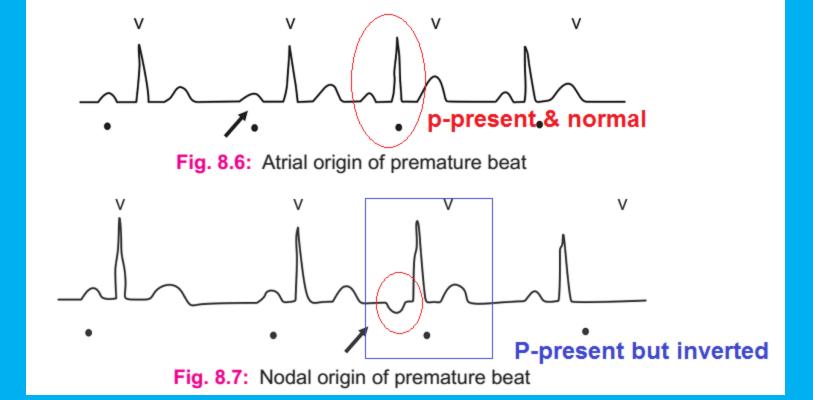
Fig. 8.2: Complete compensatory pause

- Expected occurrence of R wave.
- Expected occurrence of P wave.



	Supra-ventricular	Ventricular
QRS	Normal configuration	wide and bizarre QRS
P wave	P wave present May not visible May merge with premature T wave	No P wave
	Incomplete compensatory pause	Complete compensatory pause

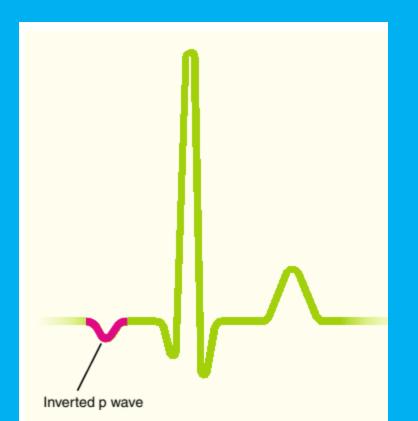


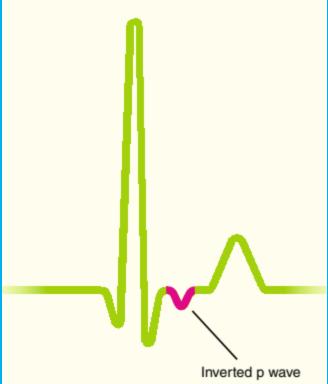


P wave
Upright P wave
Inverted P wave
just before, within or
after QRS complex.

P-R interval
short P - R
normal

Nodal Rhythm or Junctional Rhythm





1. High nodal rhythm: Inverted P wave before QRS

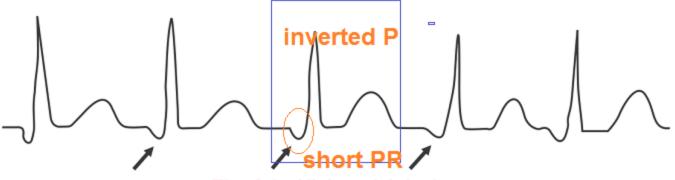
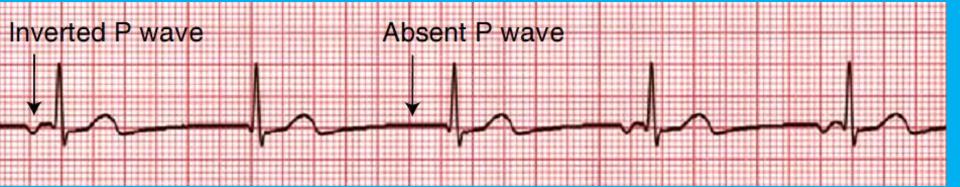


Fig. 8.9: High nodal rhythm



Mid nodal rhythm: P wave is not seen, it is buried in QRS

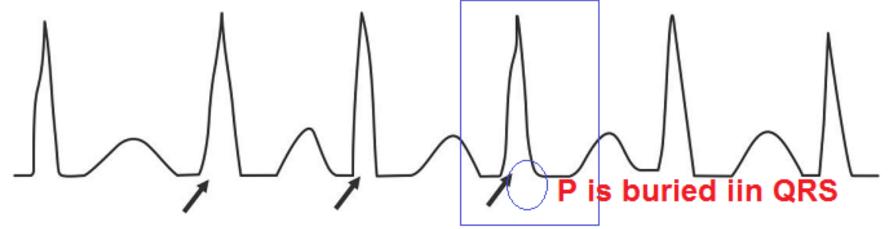
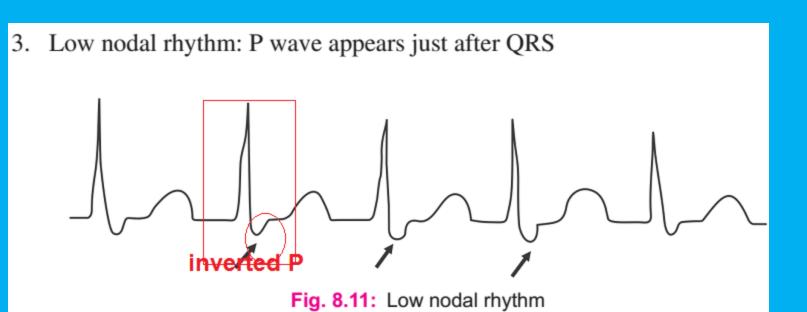
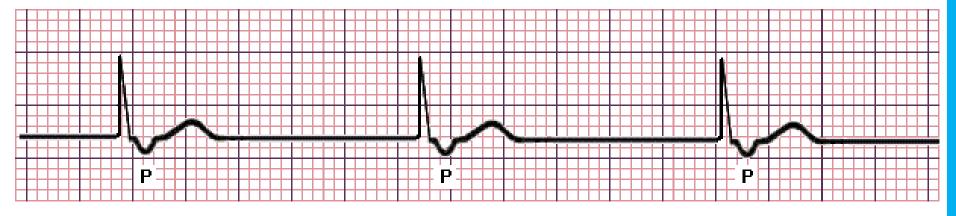
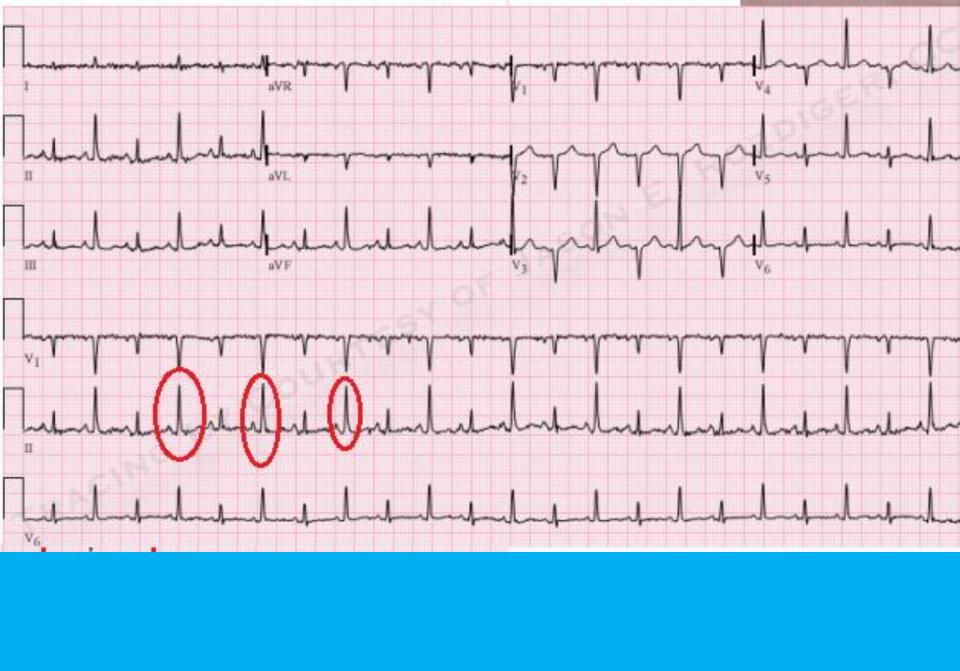


Fig. 8.10: Mid nodal rhythm



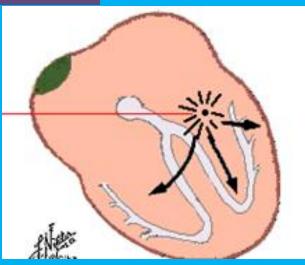
P wave, often inverted, may be buried in QRS or follow QRS. Rate slow. QRS narrow.

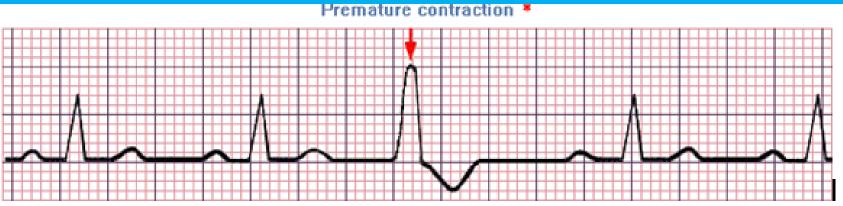




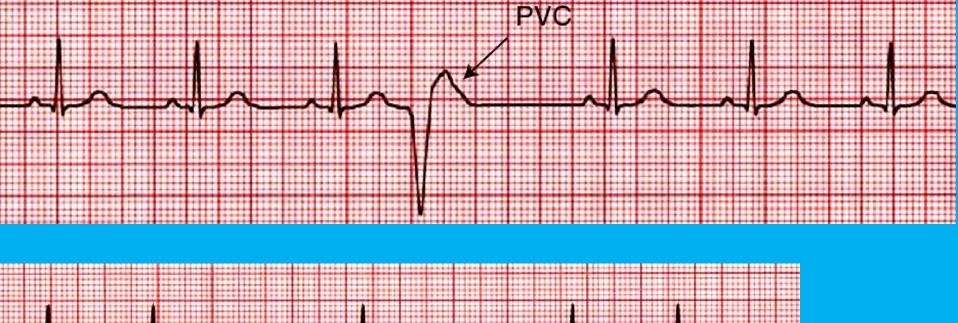
PREMATURE VENTRICULAR CONTRACTION (PVC)

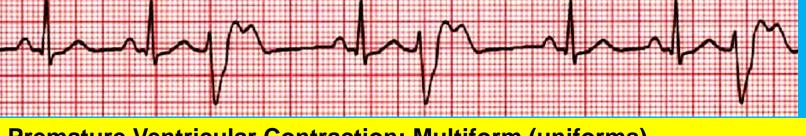
VENTRICULAR ECTOPIC





An abnormal QRS complex in normal QRS complex

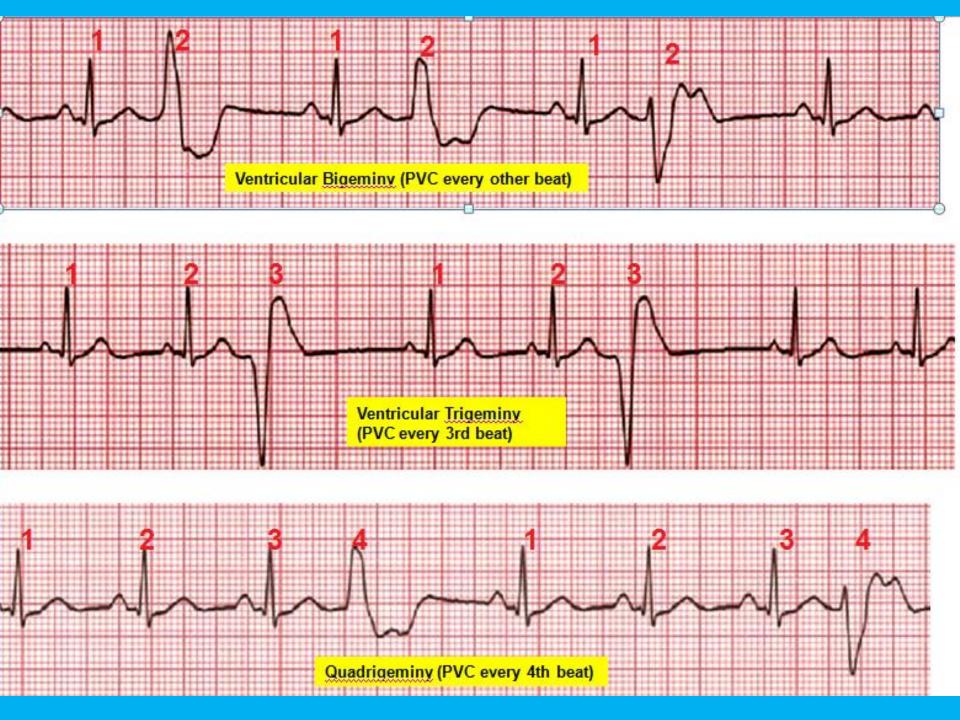


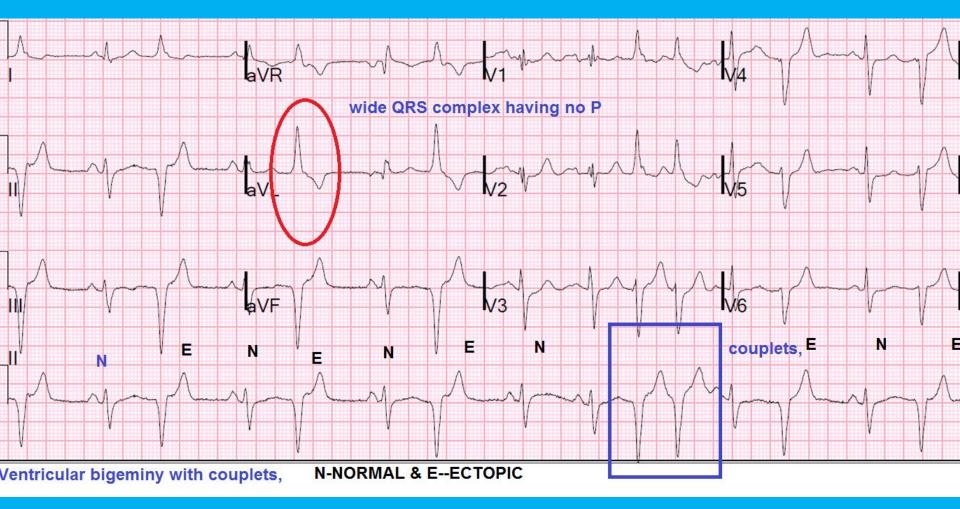


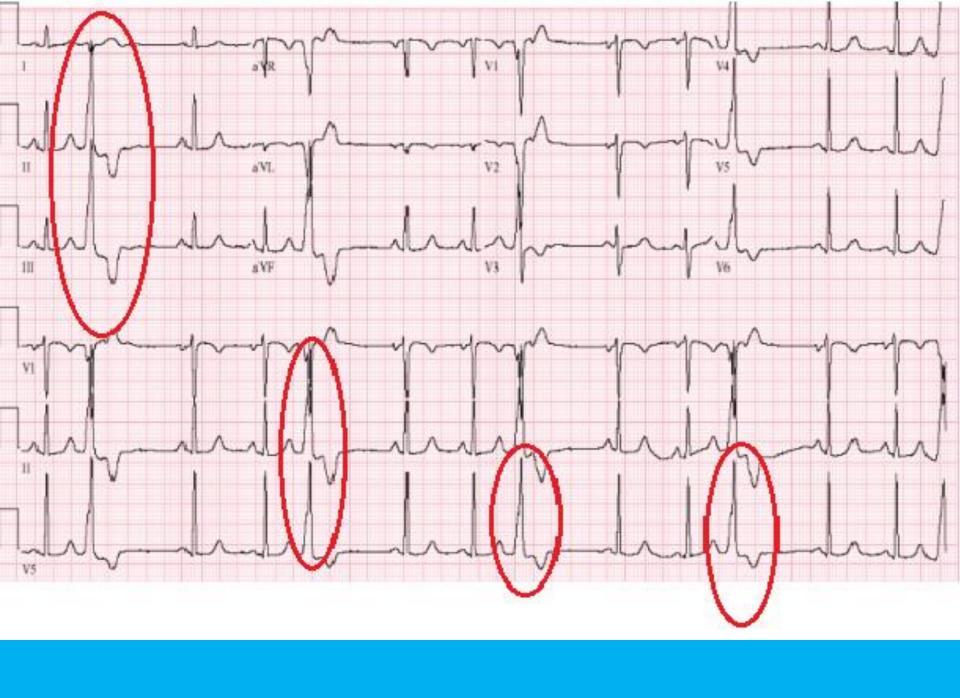
Premature Ventricular Contraction: Multiform (uniforms)

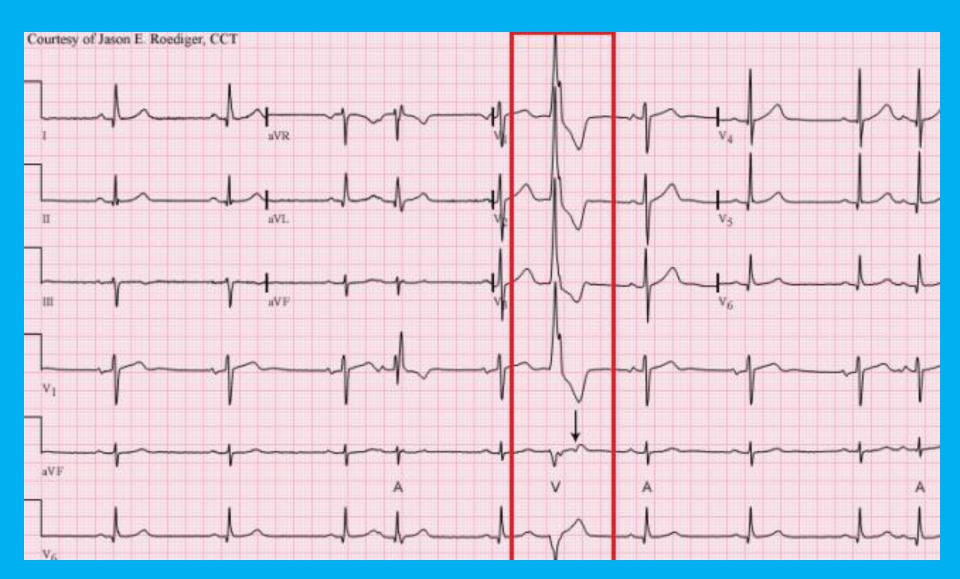


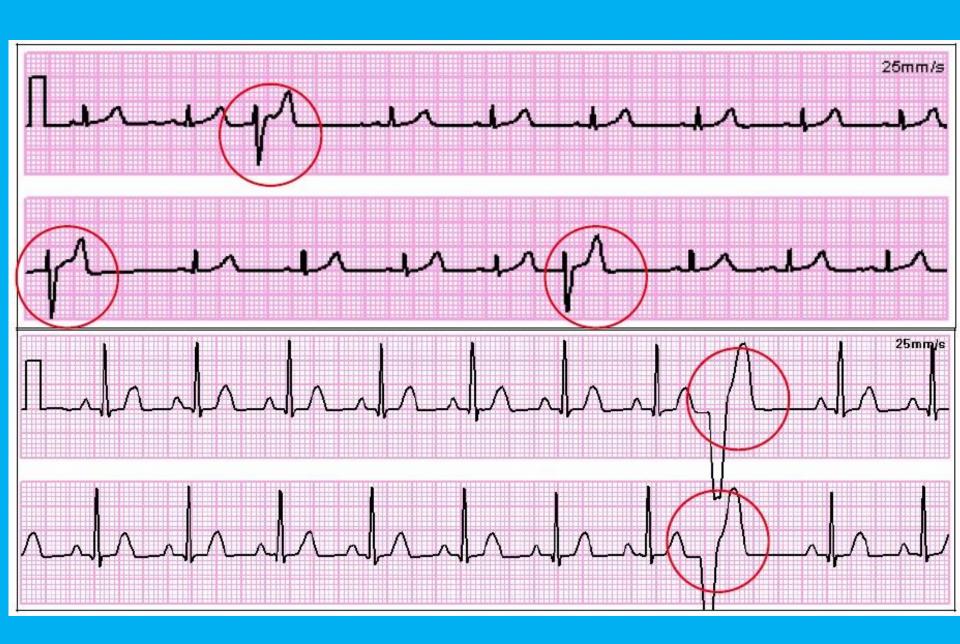
Premature Ventricular Contraction: Multiform (different forms)

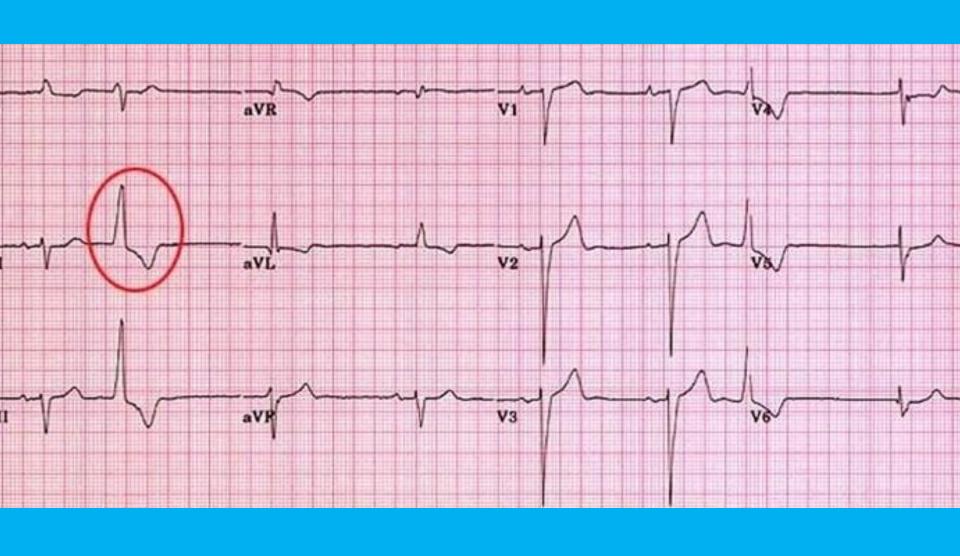




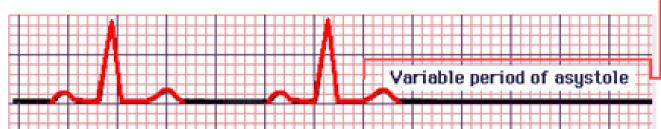








Escape Beats (Occur Late)

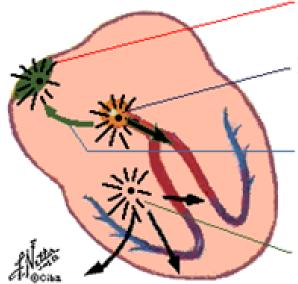


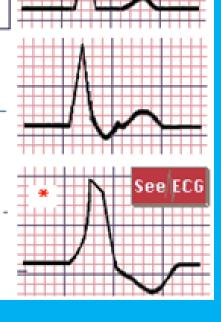
AV node takes over, usually after 1.2 to 1.6 seconds: junctional escape beat

AV node takes over, retrograde conduction: junctional escape beat

Ventricle takes over, usually after 1.8 to 2.2 seconds: ventricular escape beat

SA node resumes: sinus pause

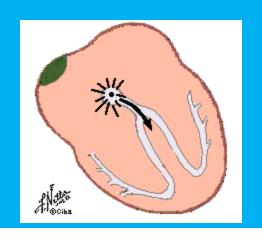


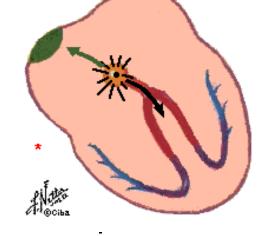


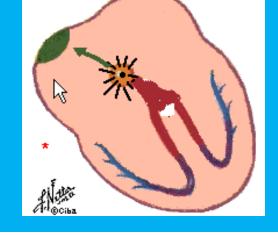
MOU. Z. Sect. 4, Ga. 9 of a

Escape beats

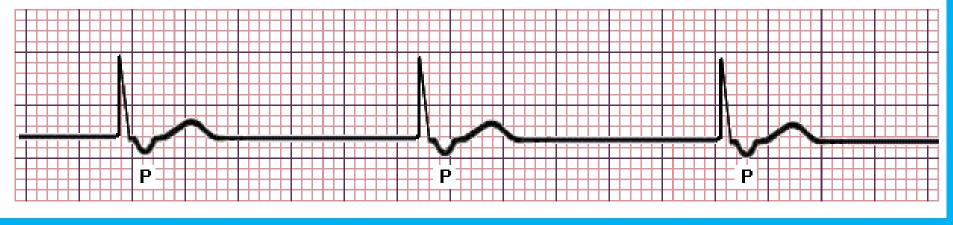
More about*



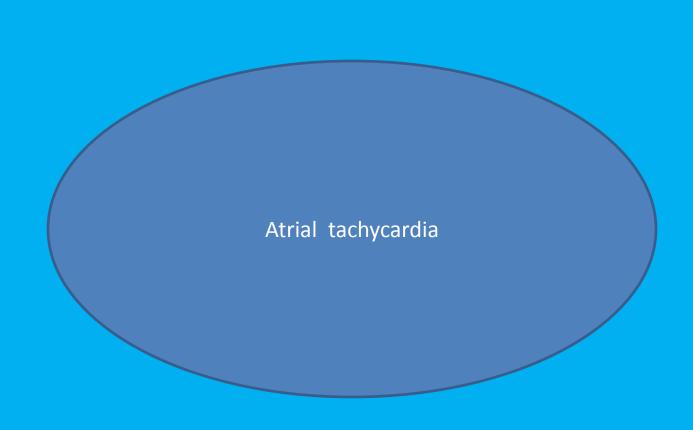




P wave, often inverted, may be buried in QRS or follow QRS. Rate slow. QRS narrow.



Retrograde or antegrade



ATRIAL TACHYCARDIA



□Rate: 150–250 bpm.

□Rhythm: Regular.

□P Waves: Normal (upright and uniform) but

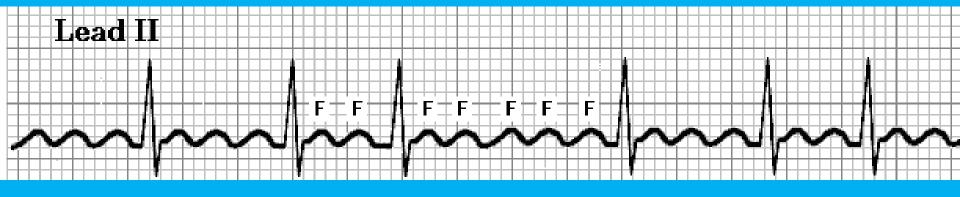
differ in shape from sinus P waves.

□PR Interval: May be short (0.12 sec) in rapid

rates.

□QRS: Normal.

ATRIAL FLUTTER





□Rate: Atrial: 250–350 bpm;

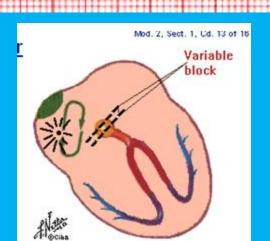
□ventricular: slow or fast

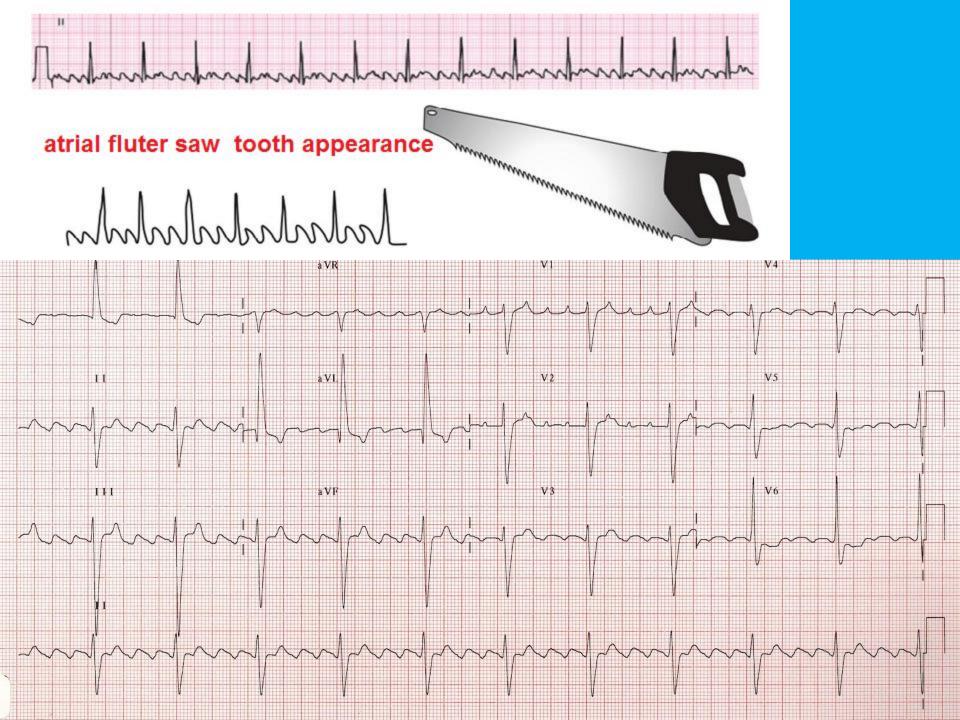
□Rhythm: Usually regular but may be variable

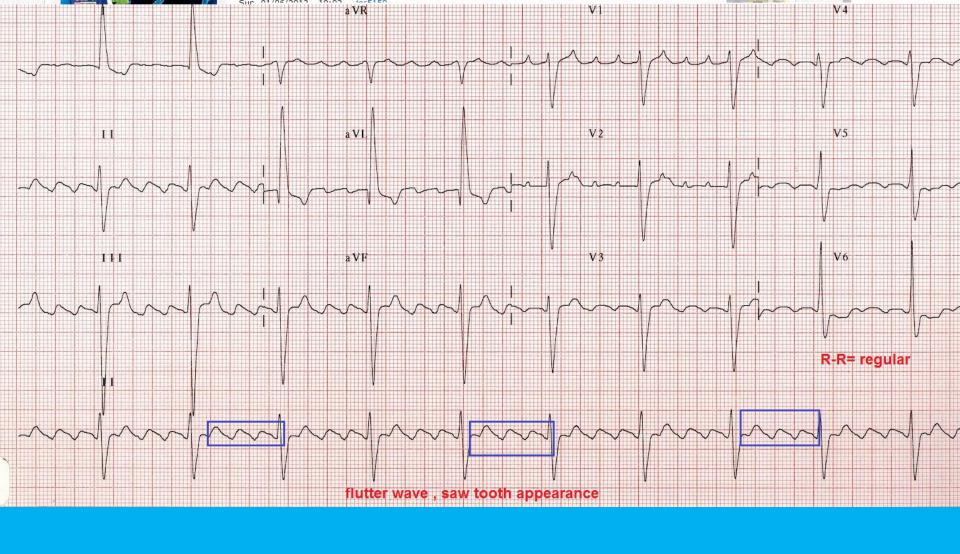
□P Waves: Flutter waves have a saw-toothed

appearance

□PR Interval: Variable

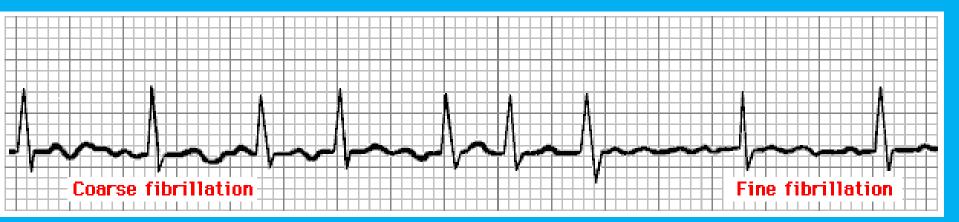






ATRIAL FIBRILLATION



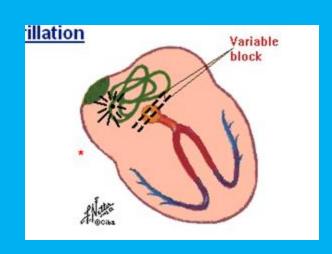


➤ Rate: Atrial: 350 bpm or greater;

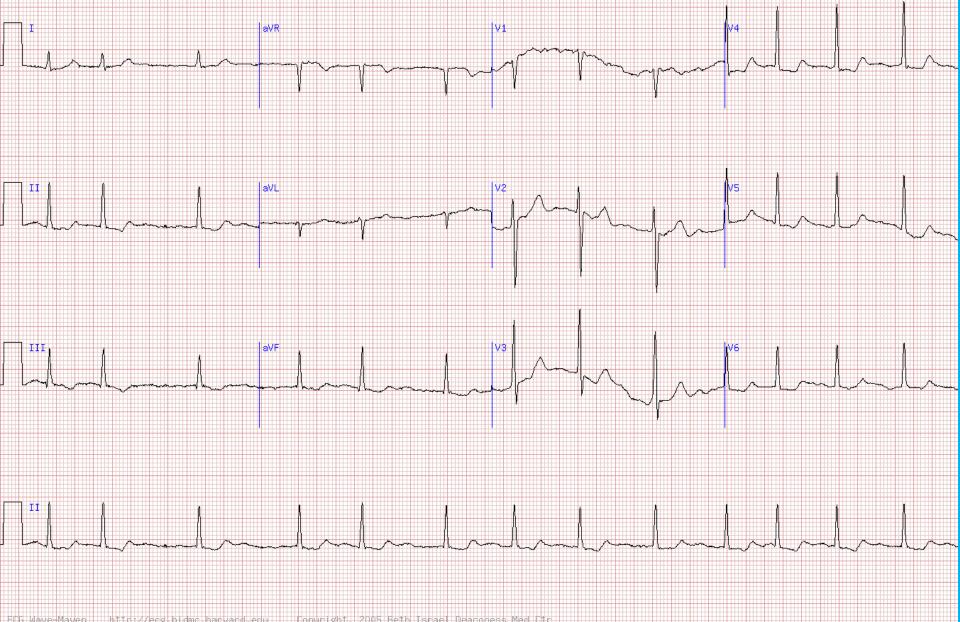
➤ Ventricular: slow or fast

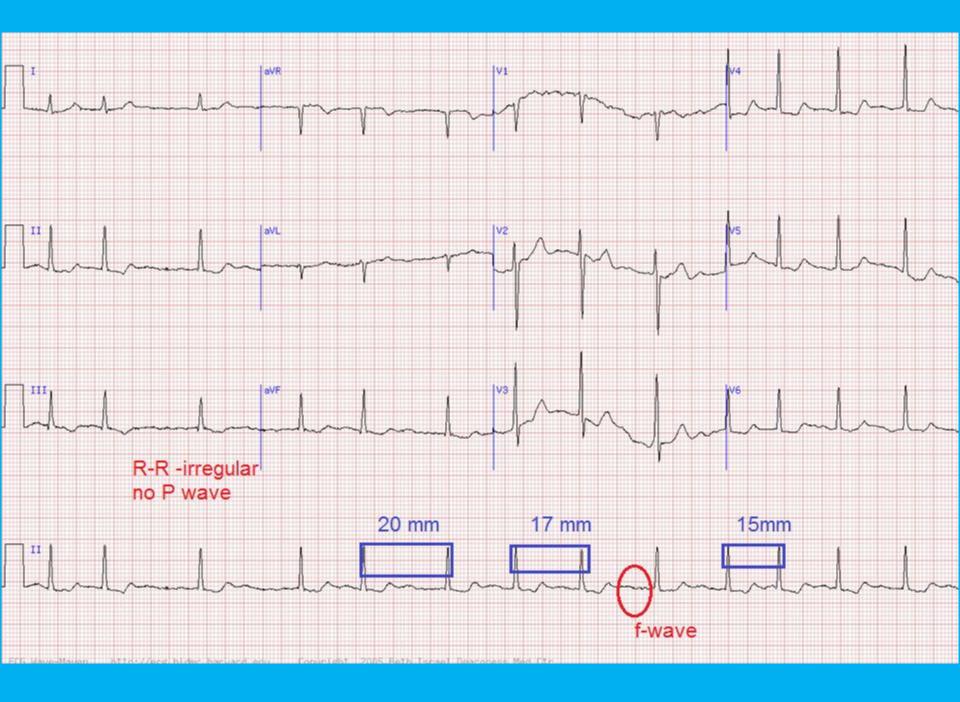
➤ Rhythm: Irregular➤ P Waves: Absent➤ PR Interval: None

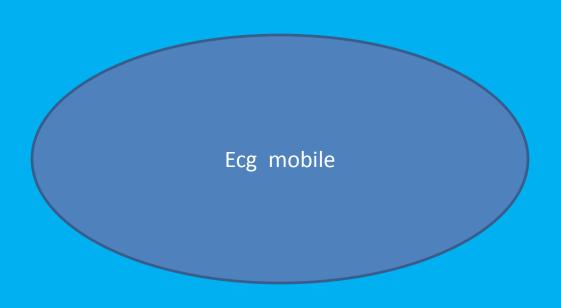
➤ QRS: Normal (0.06–0.10 sec



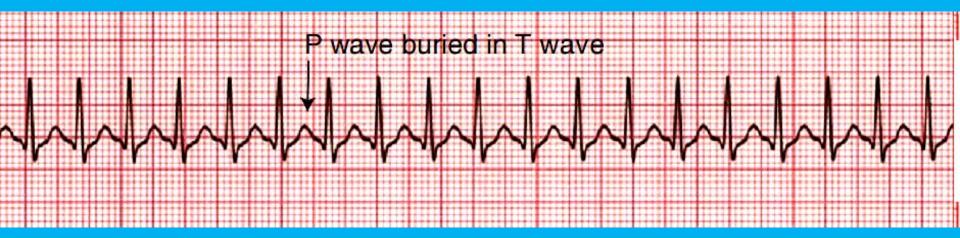












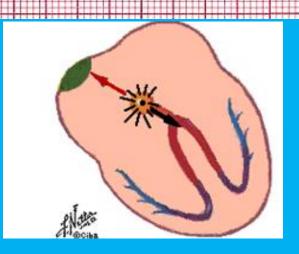


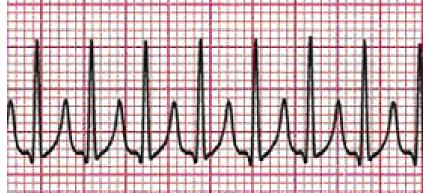
☐Rate: 150–250 bpm

☐Rhythm: Regular

□P Waves: absent (Frequently buried in preceding T waves)

□QRS: usually narrow (0.06–0.10 sec)







SVT

- **❖Pulse** = <160
- P = absent
- **♦•ORS** complex narrow

Clue to diagnosis

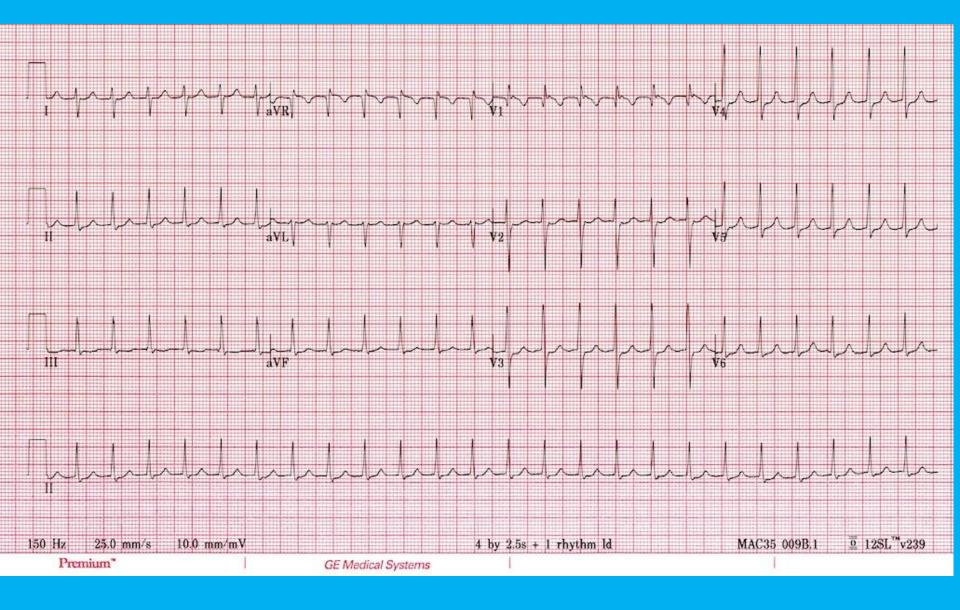
Between R_R you will find one wave that is T wave

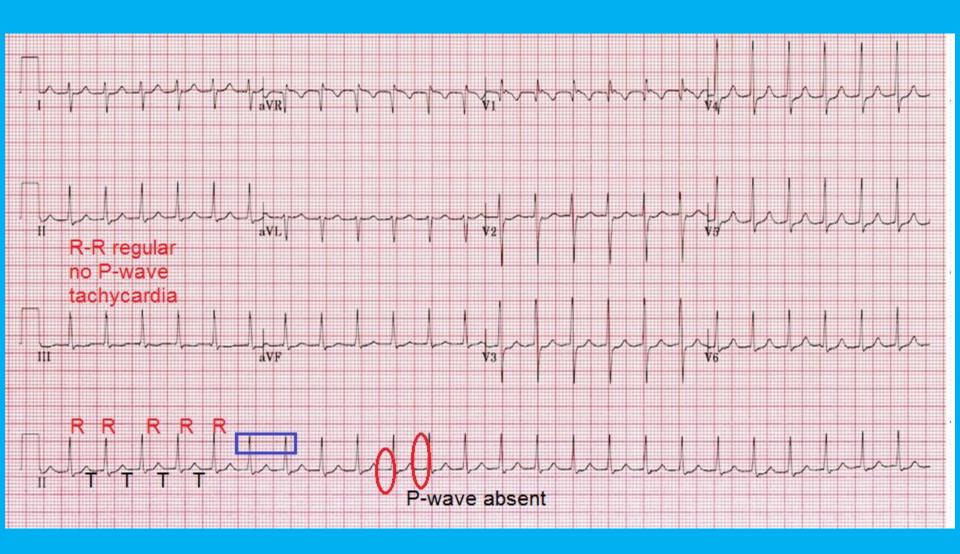
SINUS TACHYCARDIA

- **Pulse 160- 220**
- **P** present
- *****QRS complex normal

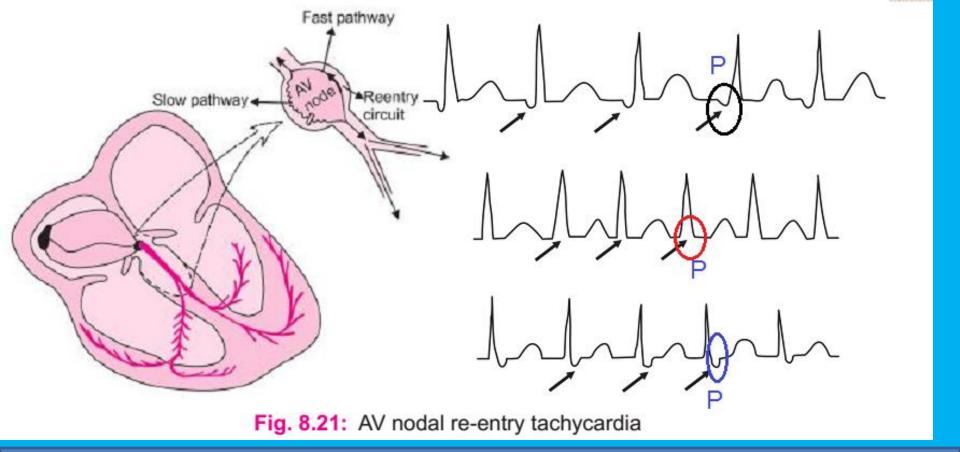
Clue to diagnosis

Between R_R you will find 2 wave that is T and P





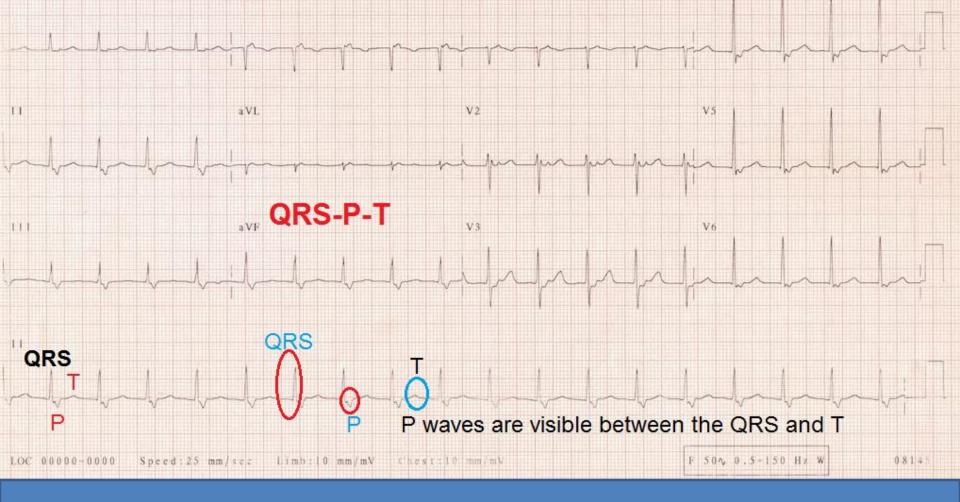
AV nodal re-entry tachycardia



AV nodal re-entry tachycardia:

Rate 140–200 beats per minute.

It is initiated by an atrial premature beat. Re-entry rhythm originates in the AV nodal area and spreads simultaneously up to the atria and down to the ventricles, as a result the P waves are usually hidden in the QRS complex because the atria and the ventricles are activated simultaneously.



Fast-Slow AVNRT (Uncommon AVNRT)

Associated with Fast AV nodal pathway for anterograde conduction and Slow AV nodal pathway for retrograde conduction.

The retrograde P wave appears after the corresponding QRS ECG

QRS -P-T complexes

P waves are visible between the QRS and T wave

AV re-entry tachycardia

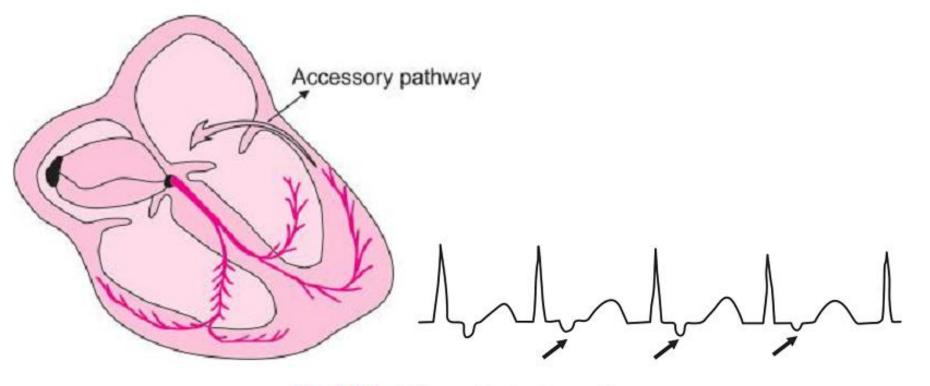
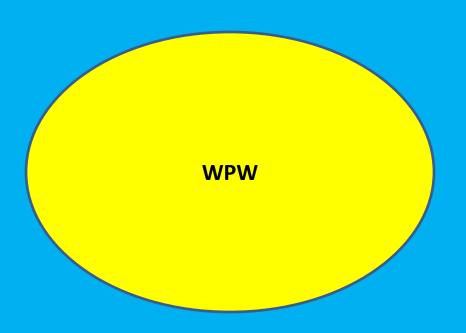
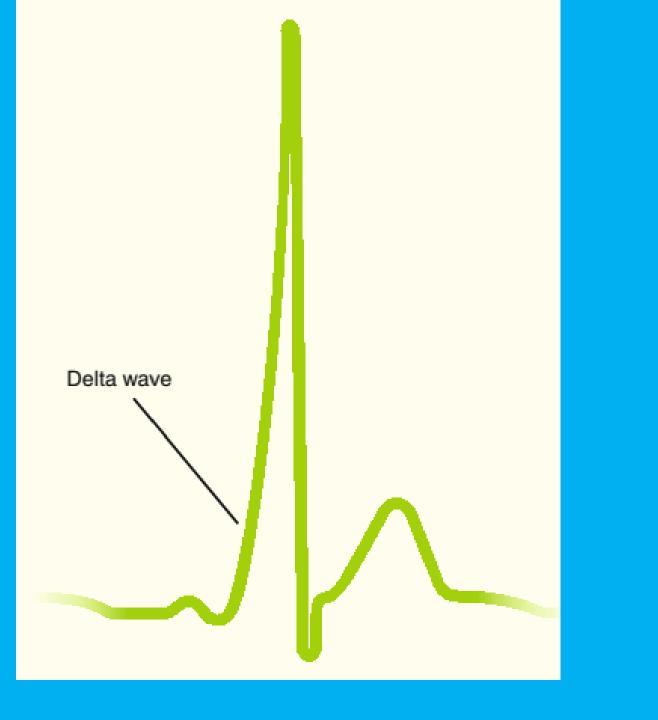


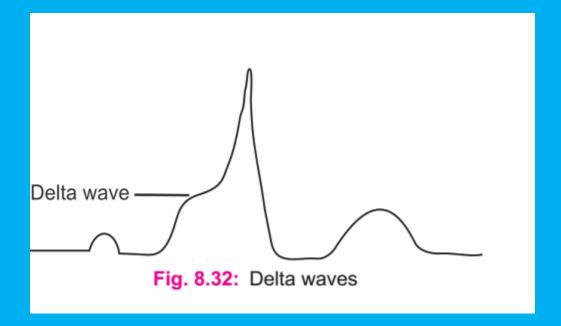
Fig. 8.22: AV re-entry tachycardia

AV re-entry tachycardia:

It is because of bypass tract (accessory pathway), i.e. an abnormal cardiac muscle connects the atria and the ventricles bypassing the AV node. From here the impulse passes down through normal conducting system (i.e. AV node bundle of His) into the ventricles, recycles rapidly by the bypass tract to the atria.







Criteria

WPW produces the following characteristic triad of fiding on ECG:

- Short P-R interval (less than 0.12 seconds)
- Wide QRS (more than 0.10 seconds)
- Delta waves

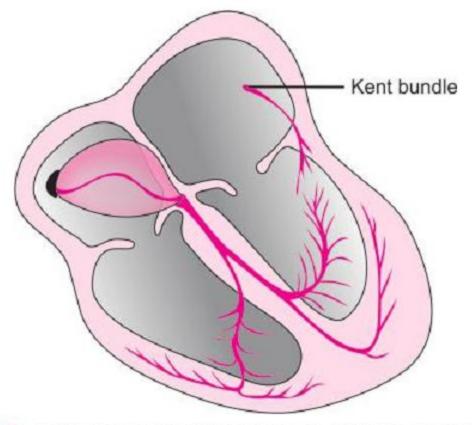
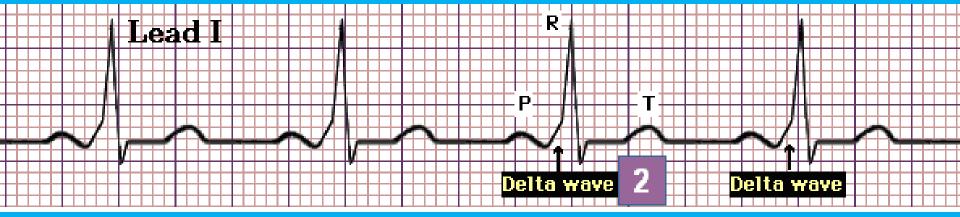


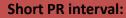
Fig. 8.33: WPW have a tract that bypasses AV node (Kent bundle)

Patient with WPW have a tract that bypasses AV node which is known as Kent bundle. In this condition, when impulse travels down through the atria it reaches the Kent bundle and AV node simultaneously. The impulse travels down the AV node and is met by normal physiological block. The impulse also travels down the Kent bundle, does not meet any block and so begins to spread through the ventricular myocardium. This progression is slow and gives a wide pattern on the ECG.

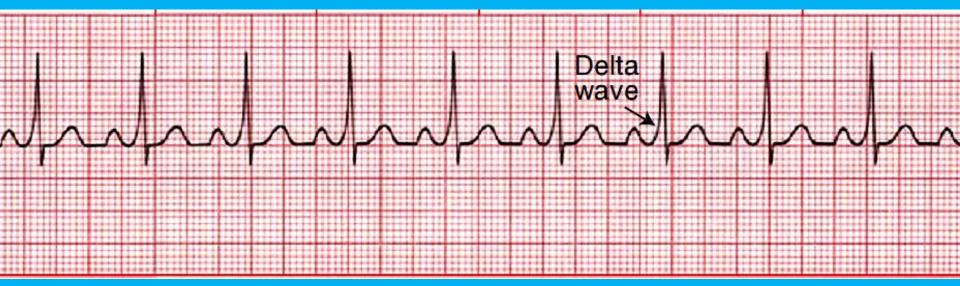


WPW Type A and B

- There are two types of WPW, type A and B.
- Type A QRS complexes are positively deflected in V1
- Type B QRS complexes are negatively deflected in V1



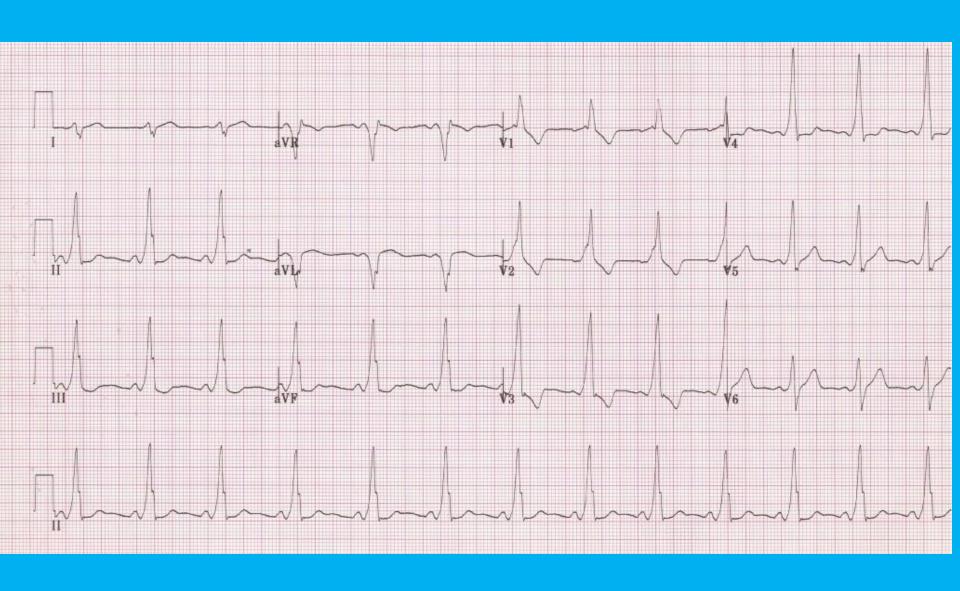
- WPW syndrome
- Nodal rhythm

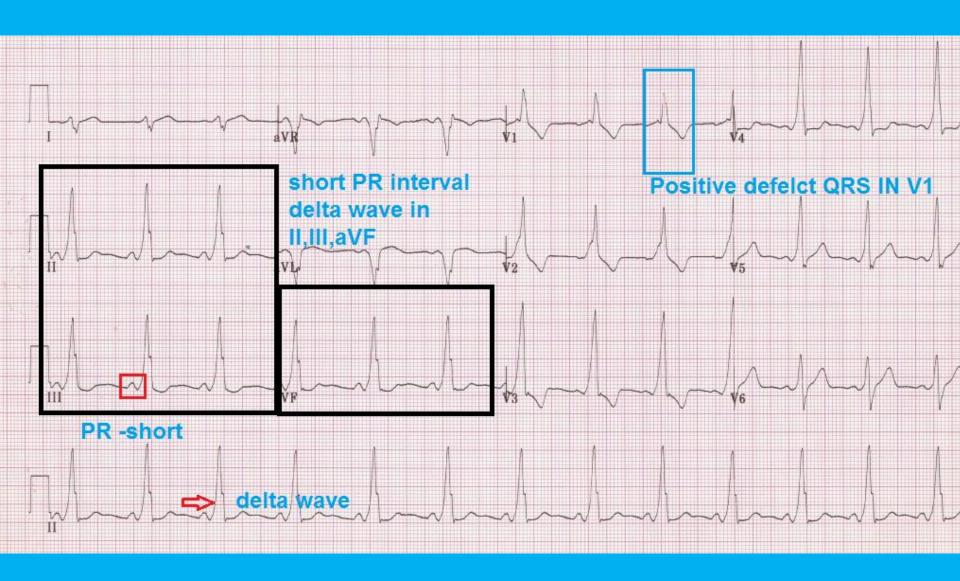


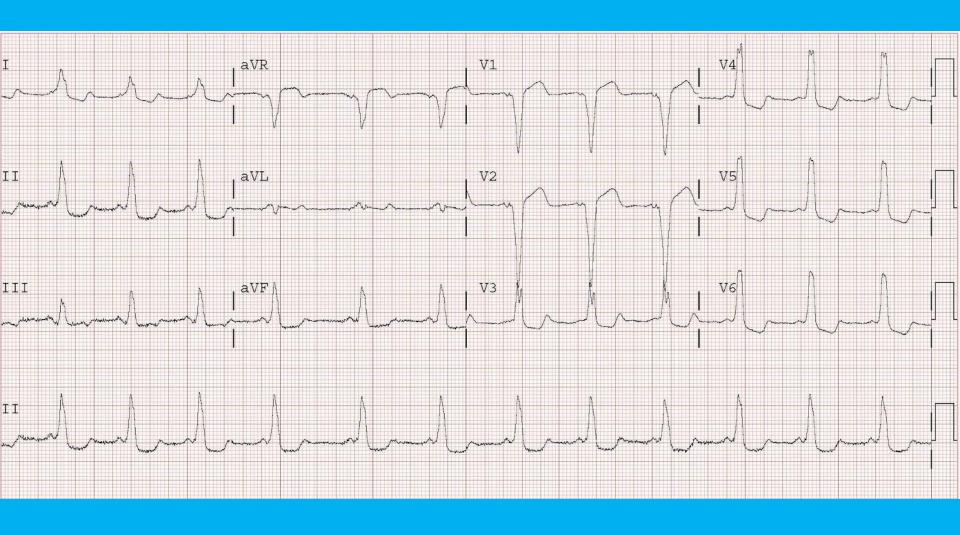
PR Interval: Short (< 0.12 sec)

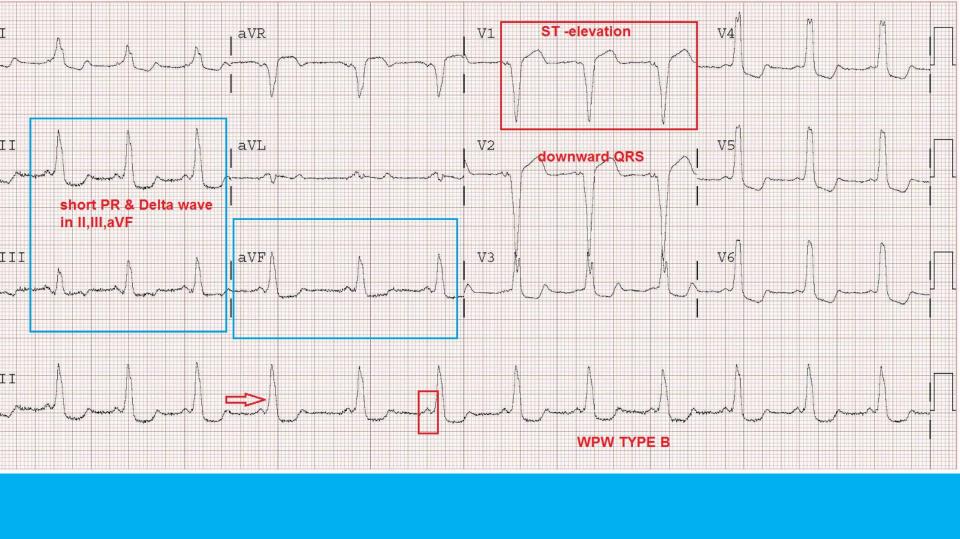
QRS: Narrow and **Delta wave** present

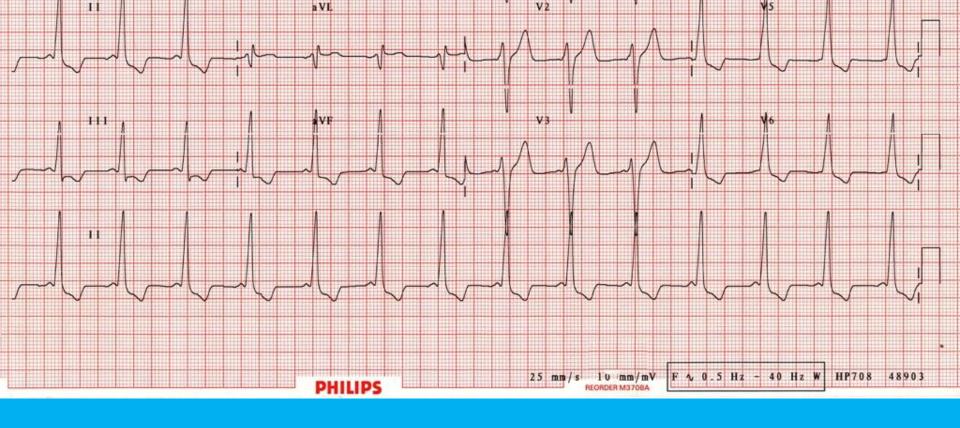
- >WPW there is accessory conduction pathway between the atria and the ventricles.
- ➤ So impulses are rapidly conducted to the ventricles. So short PR interval and there is slurring of the initial portion of the QRS called the delta wave

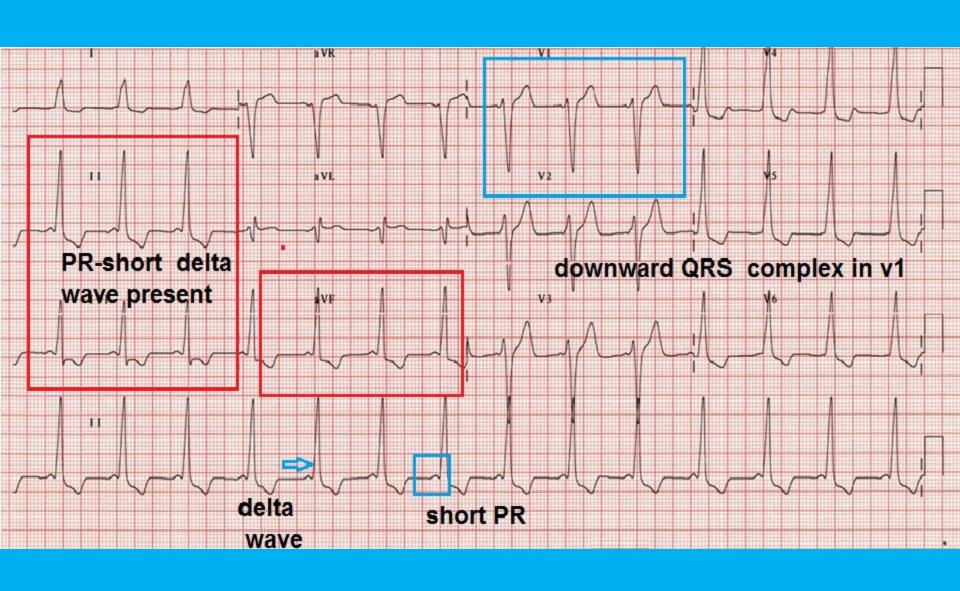


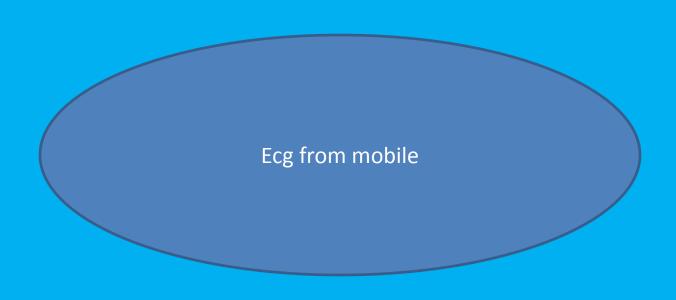




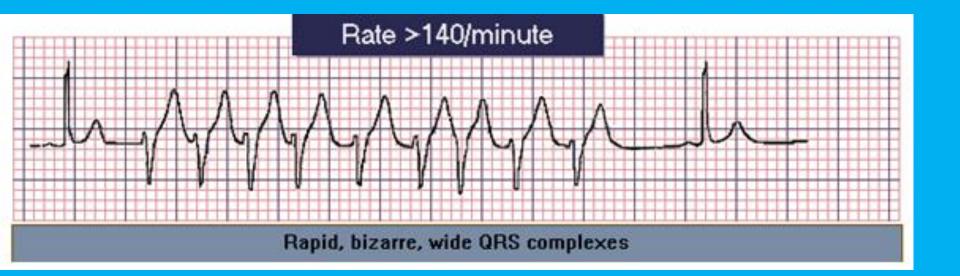








VENTRICULAR TACHYCARDIA (VT)



Rate: 100-250 bpm

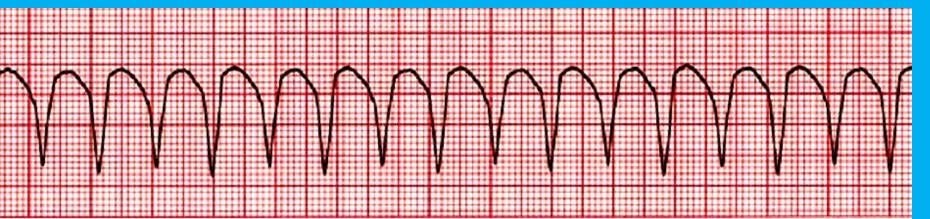
Rhythm: Regular

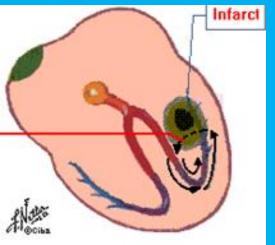
P Waves: absent

QRS: Wide bizarre appearance

VENTRICULAR TACHYCARDIA (VT)







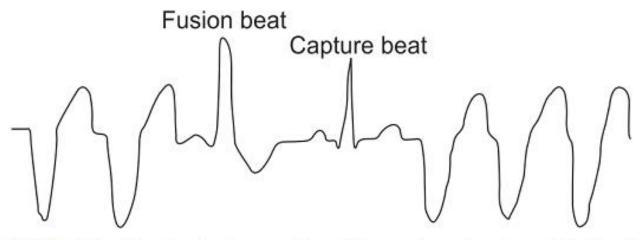


Fig. 8.27: Ventricular tachycardia with capture beat and fusion beat

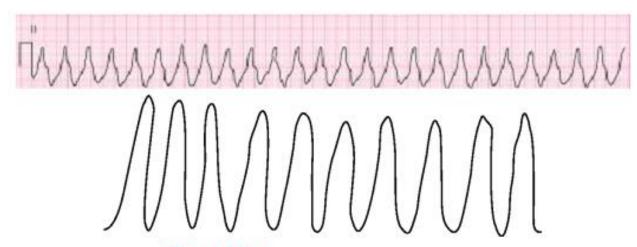


Fig. 8.28: Ventricular tachycardia

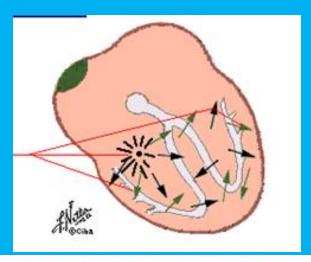
Capture : Appearance of normal QRS complex in the middle of ventricular beat tachycardia

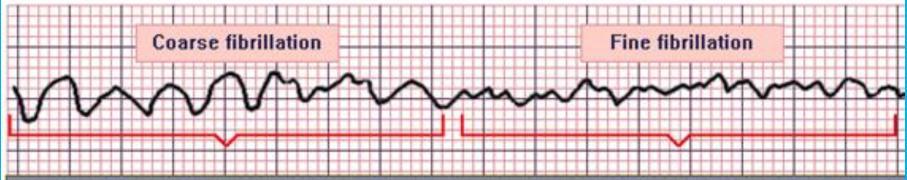
Fusion beat: This type of complex is caused by two pacemakers, SA node and ventricular pacer. The result is hybrid of fusion complex, which is a complex with some features of both.





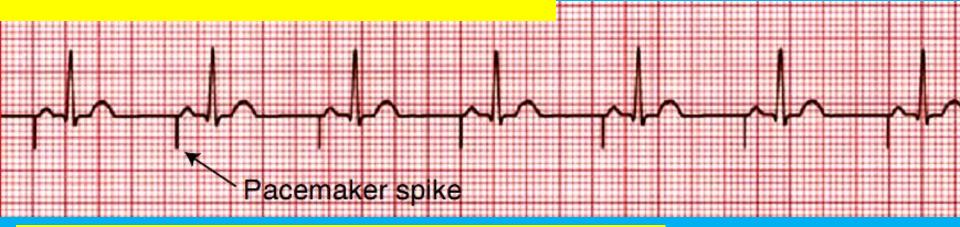
VENTRICULAR FIBRILLATION (VF)



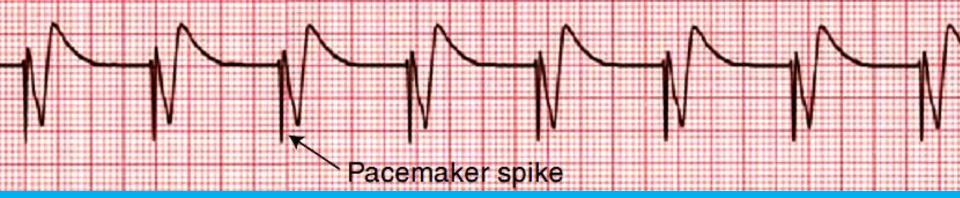


Fibrillation may be associated with either coarse or fine chaotic undulations of the ECG baseline, but no true QRS complexes. Indeterminate rate.

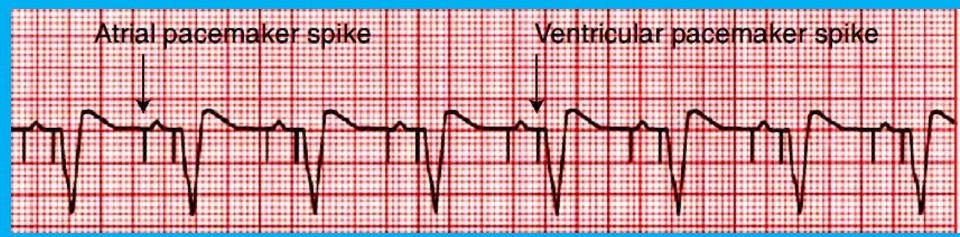


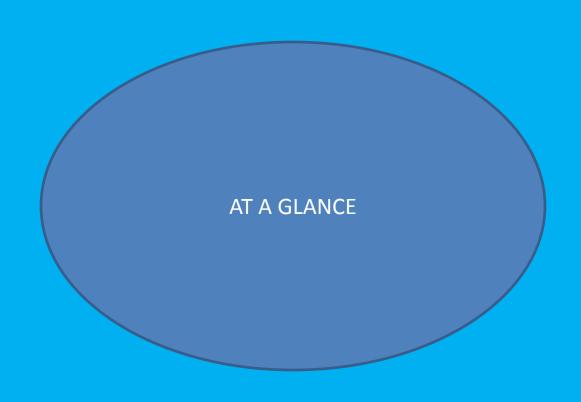


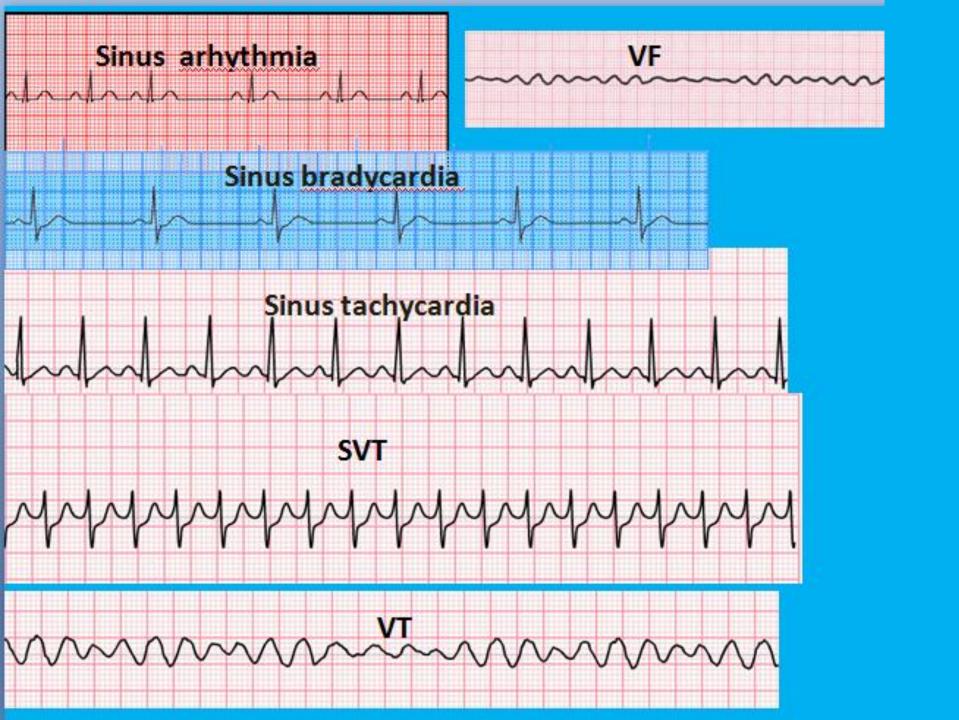


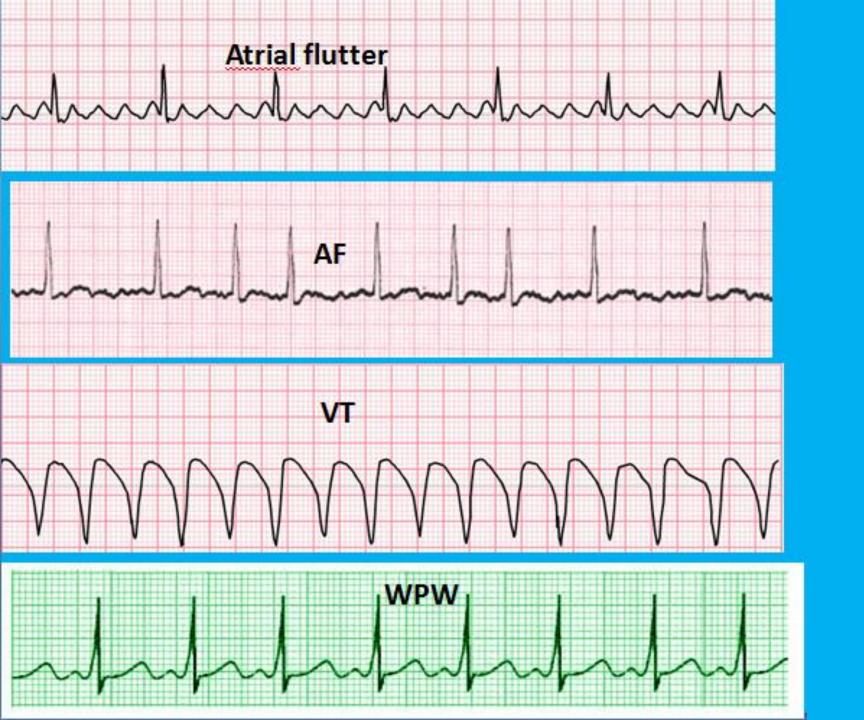


Dual-Chamber Pacemaker Rhythm—Atrial and Ventricular









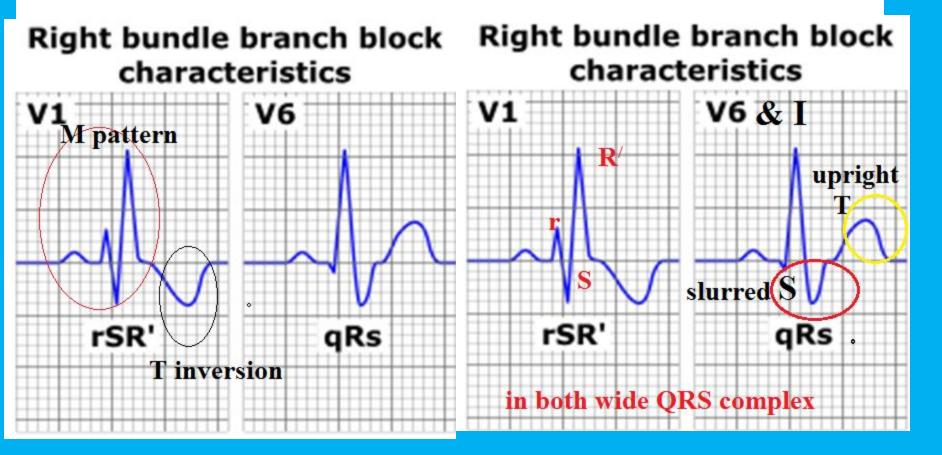
BUNDLE BRANCH BLOCK

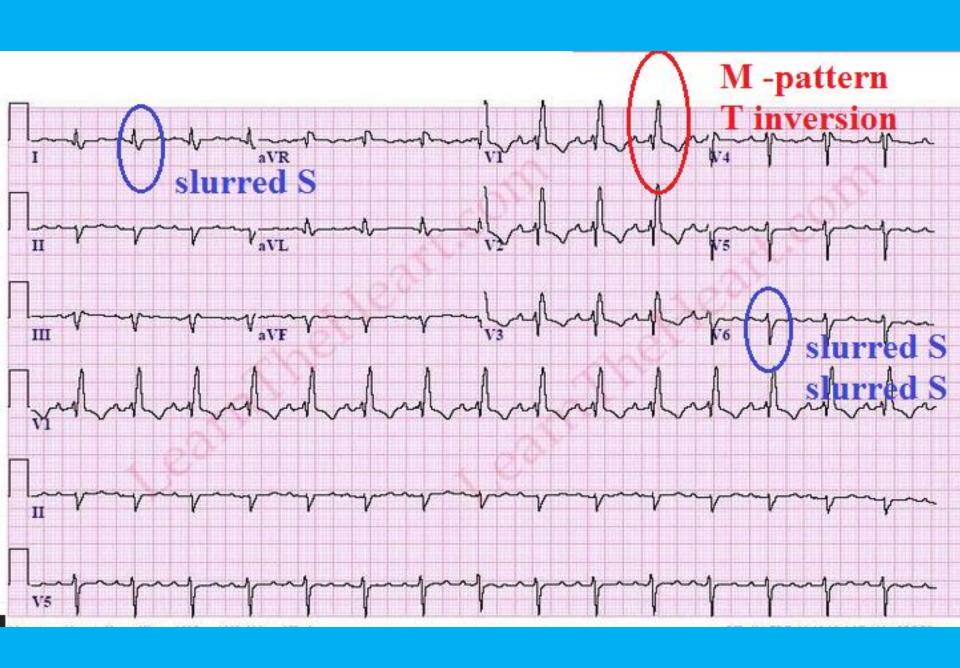
RBBB

Right Bundle Branch Block

Criteria

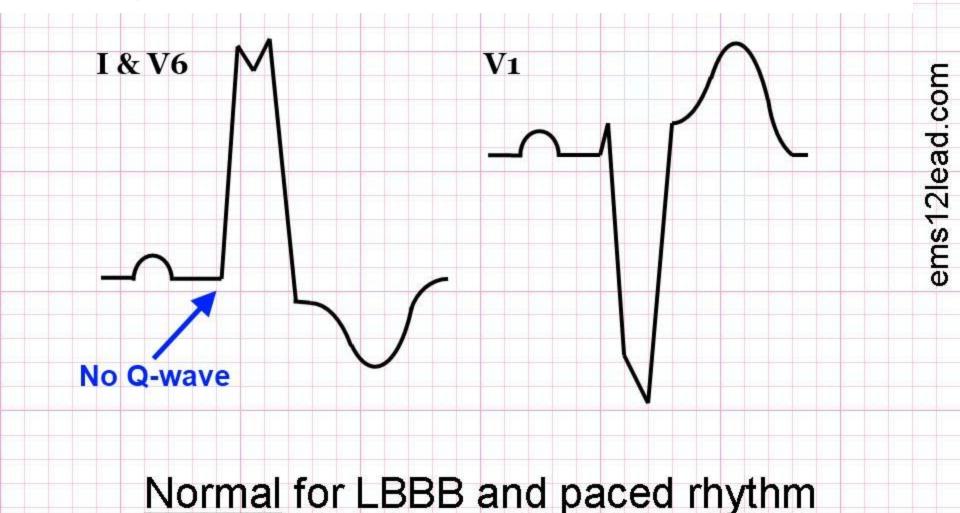
- i. Wide QRS complex
- ii. RSR pattern or rabbit ear pattern in V1
- iii. Broad and slurred S wave in leads I and V6
- iv. Right axis deviation may be present Slurred S wave in leads I and V6 are the major criteria that have to be looked.

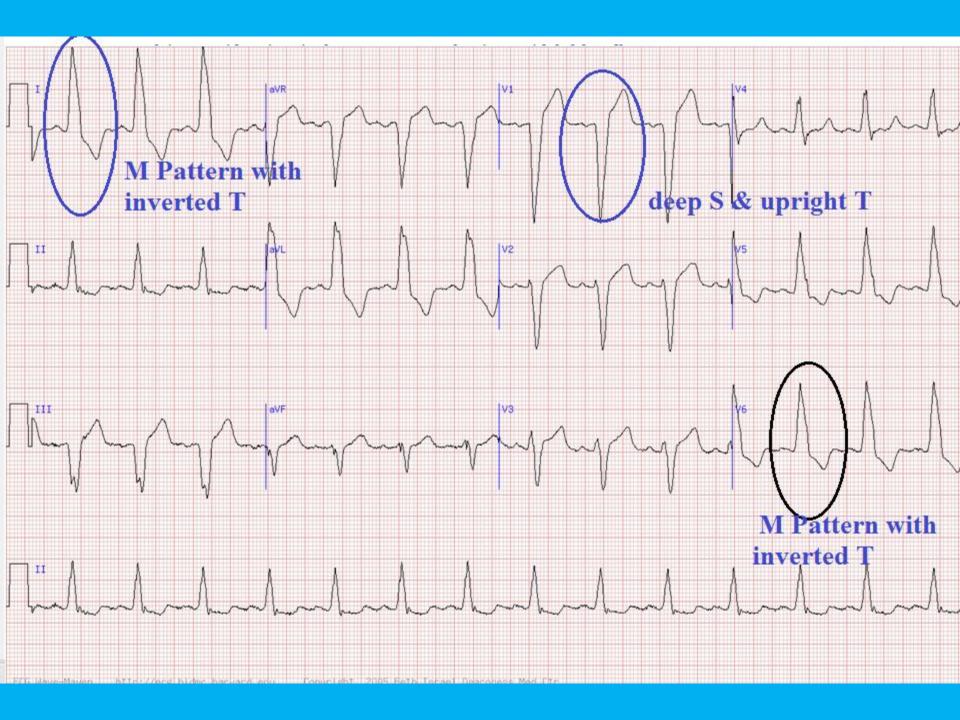


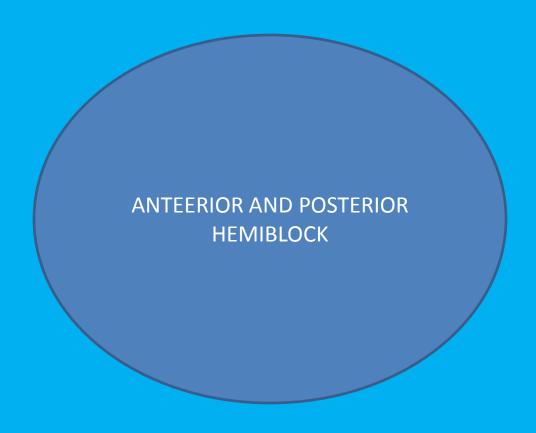




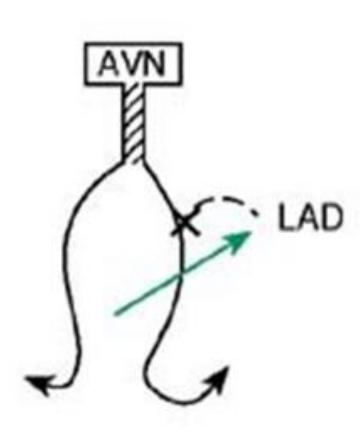
- i. Wide QRS complex with duration of > 0.12 s (> 3 mm)
- ii. Deep and broad S wave in V1 with no R wave.
- iii. Broad slurred R wave or RR' pattern without a Q wave in leads I and V6.
- iv. Always associated with left axis deviation.



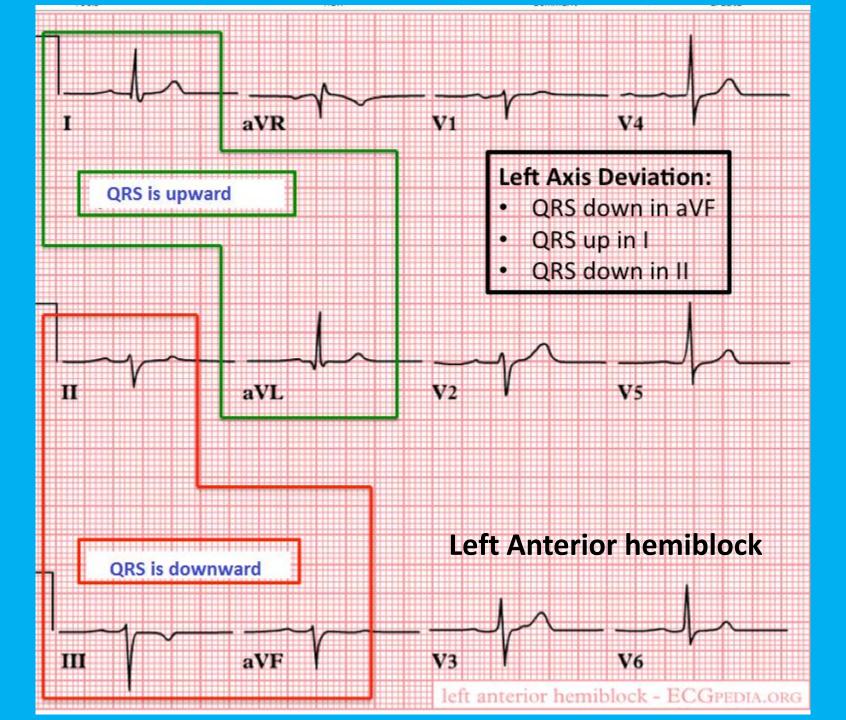


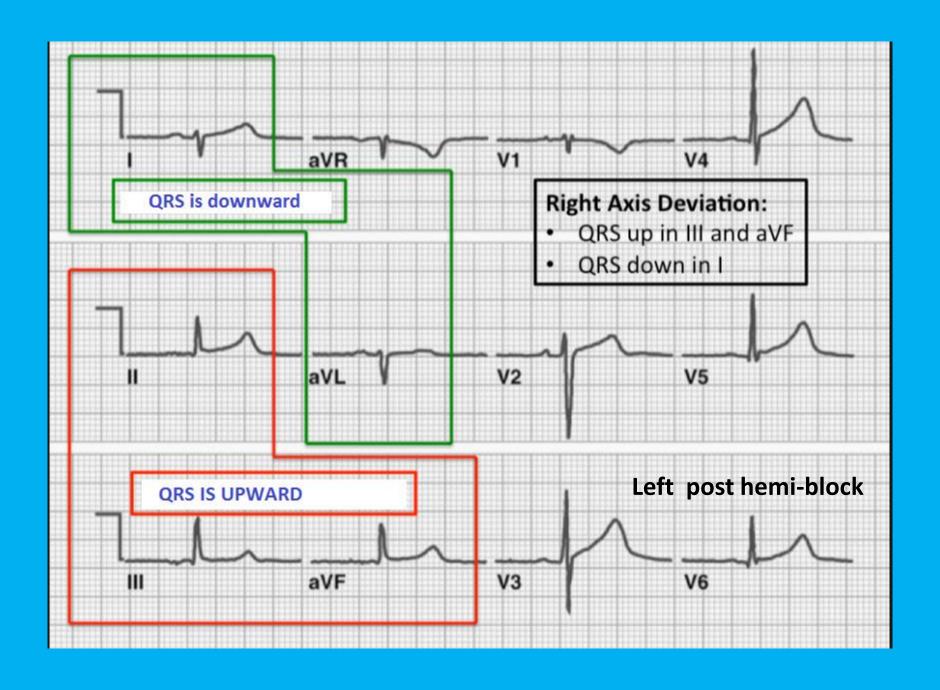


LEFT ANT. HEMIBLOCK

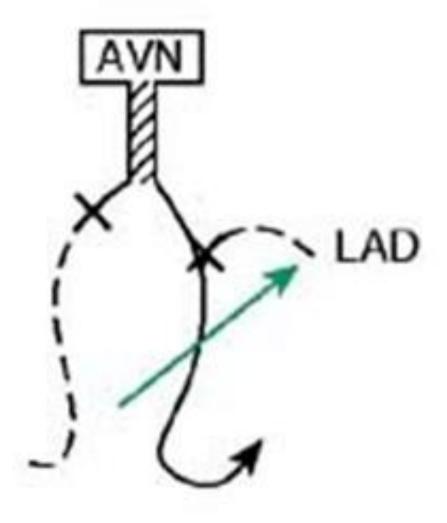


 Left anterior hemiblock → left axis deviation

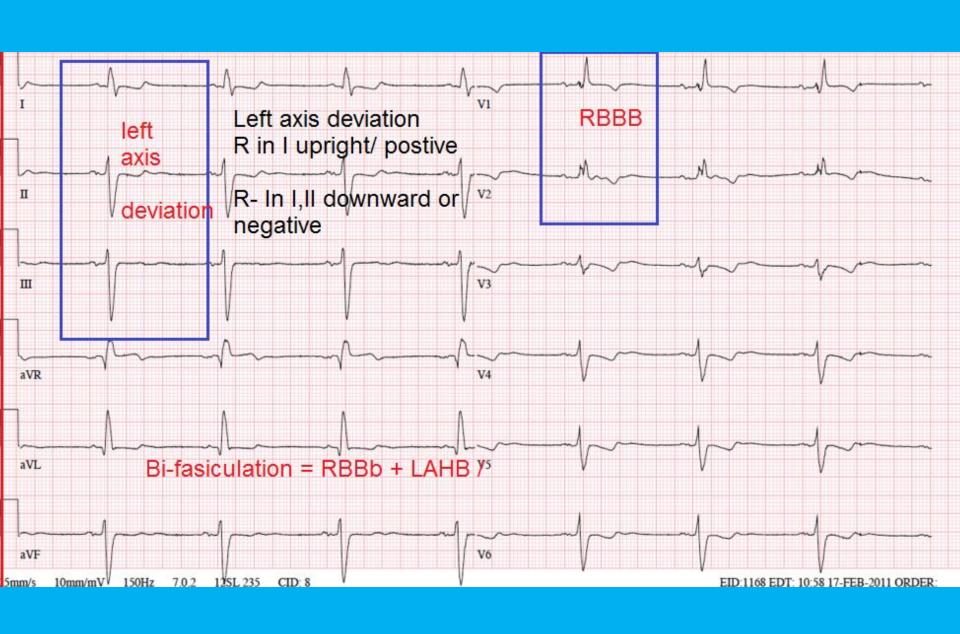


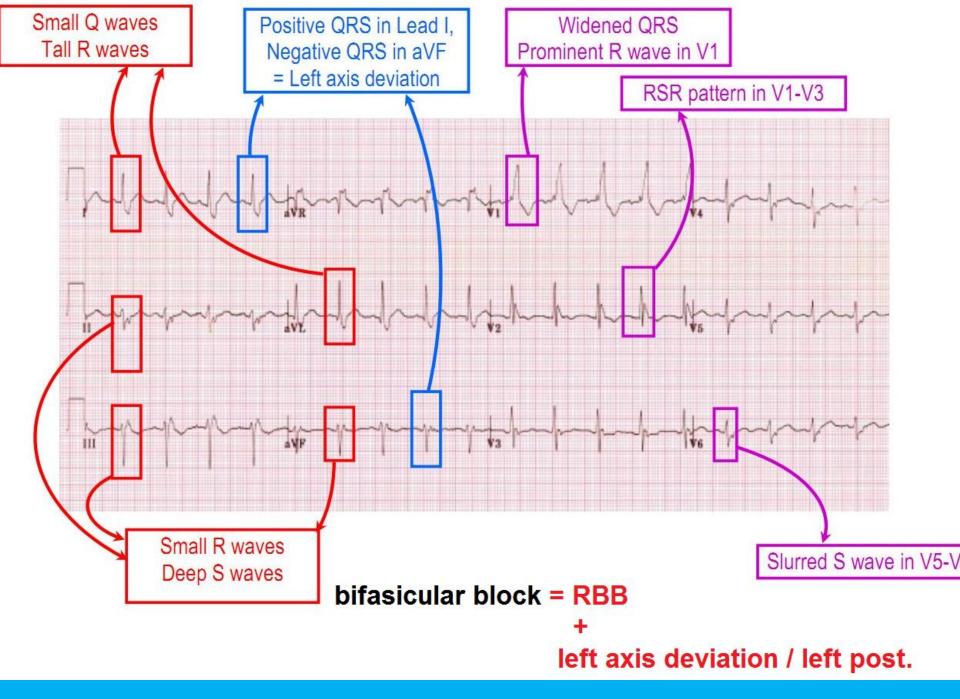


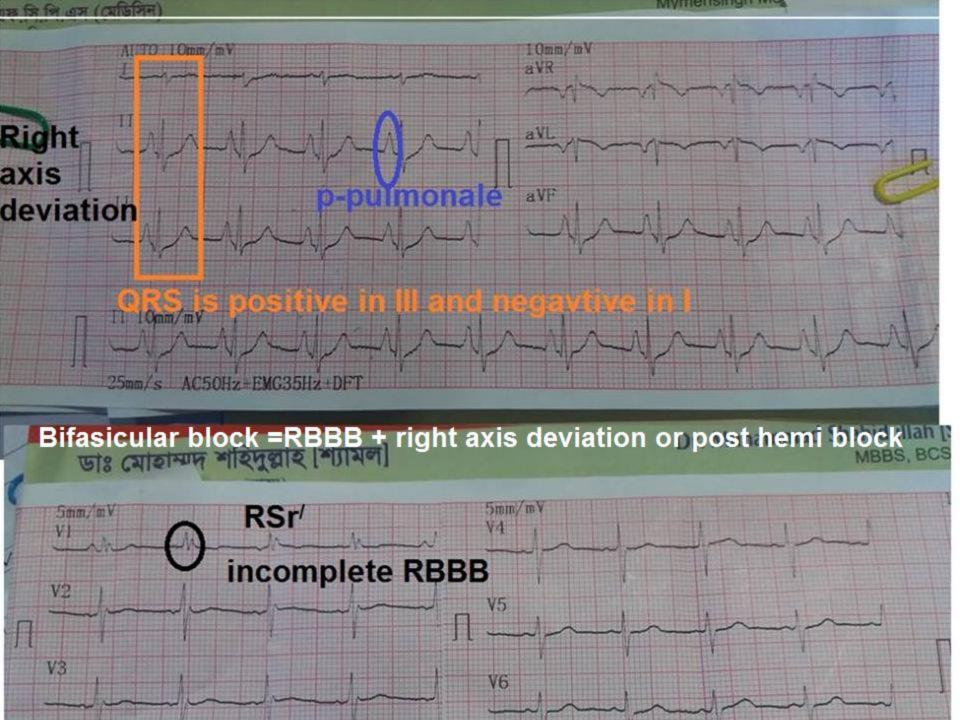
BI-FASICULAR BLOCK

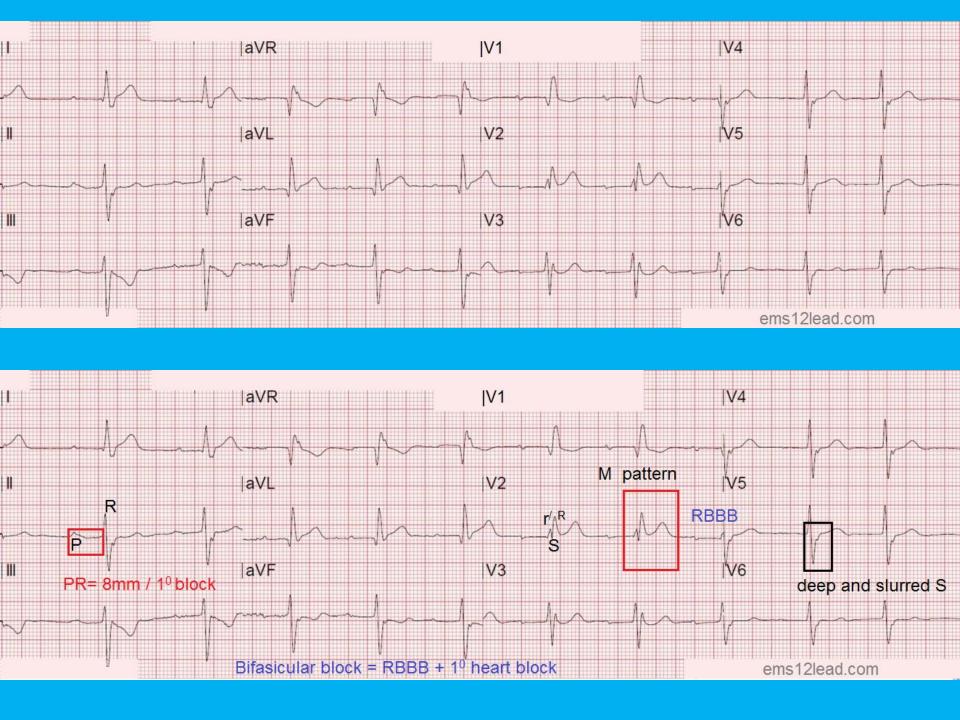


 RBBB and left anterior hemiblock → left axis deviation

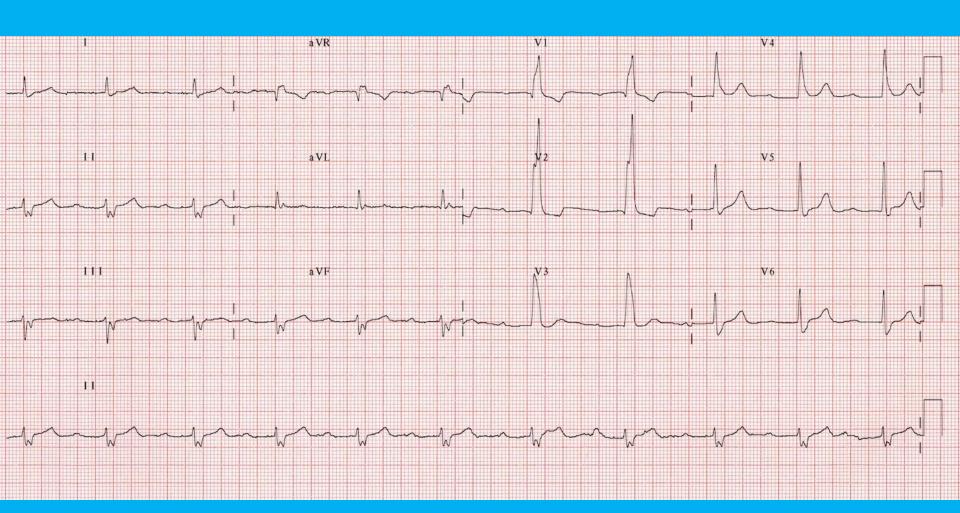


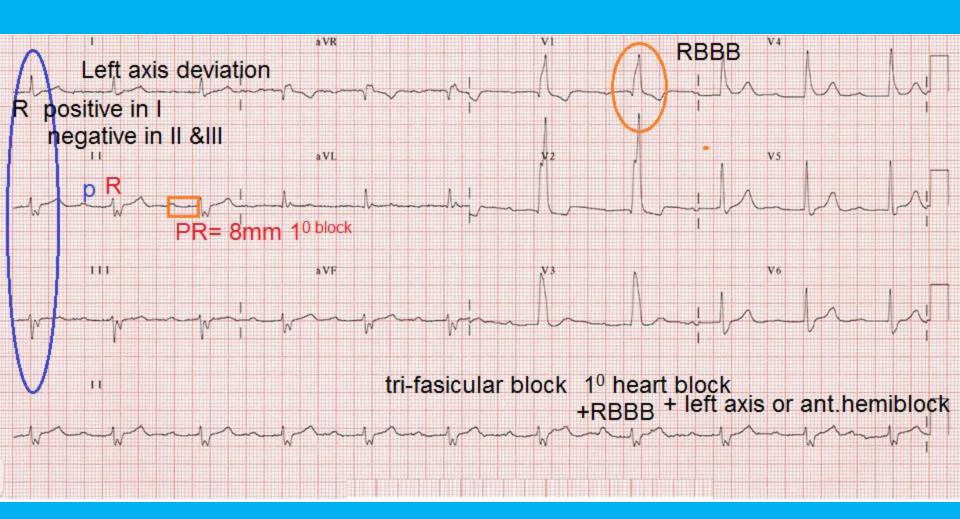


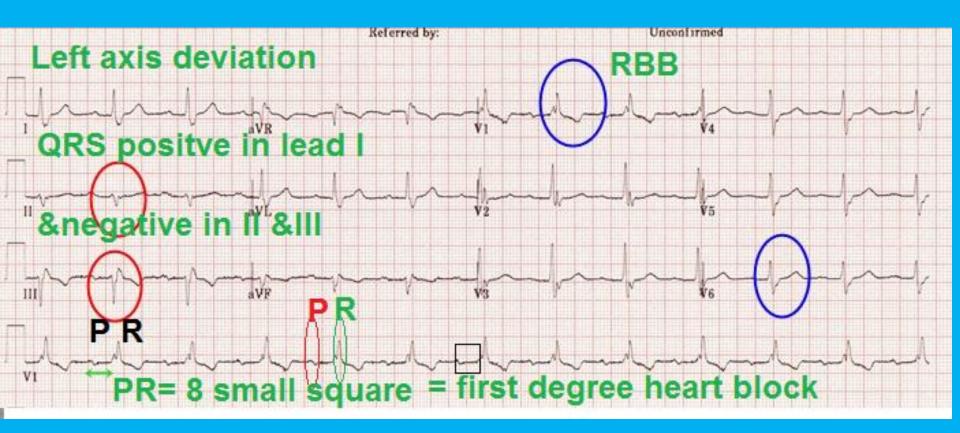


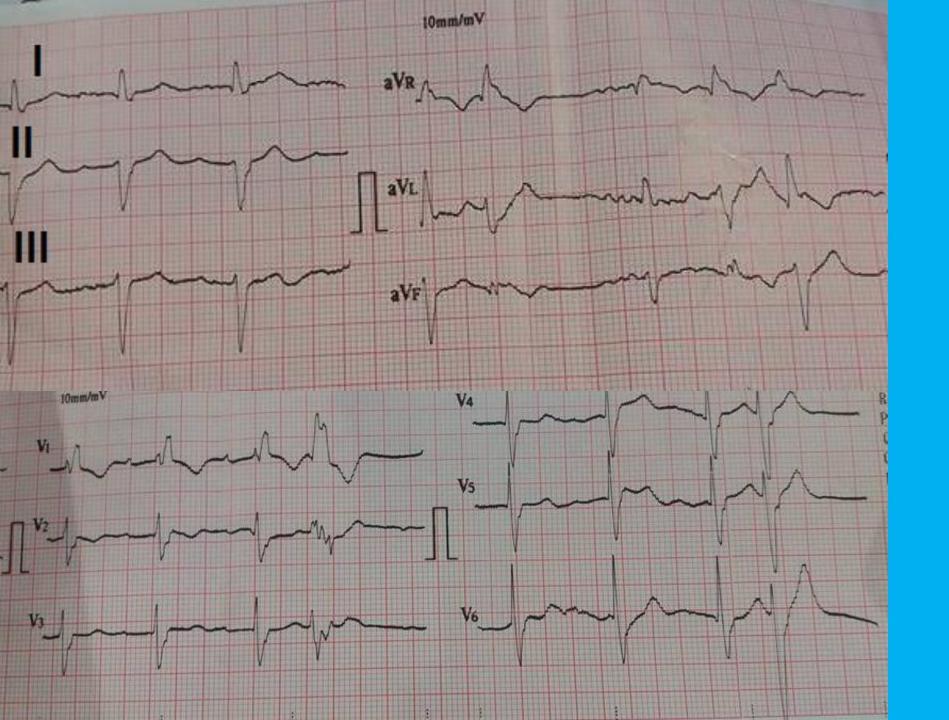


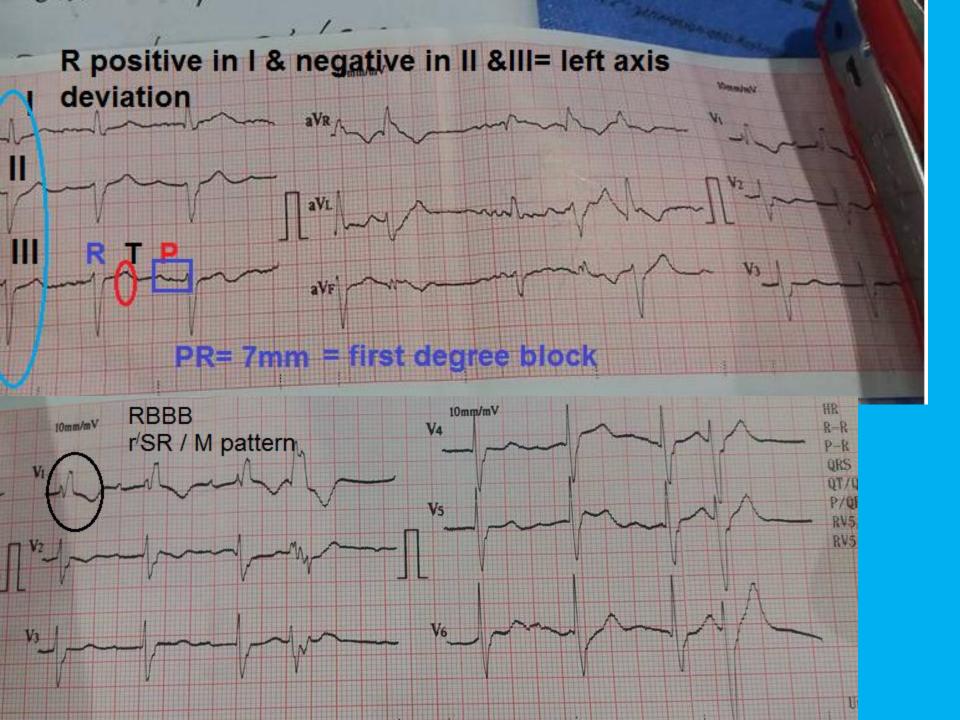
Tri-fascicular block

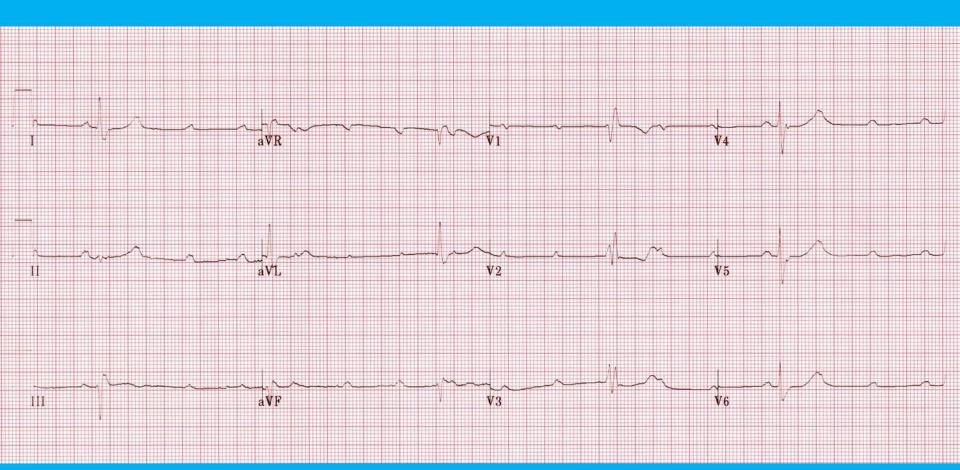


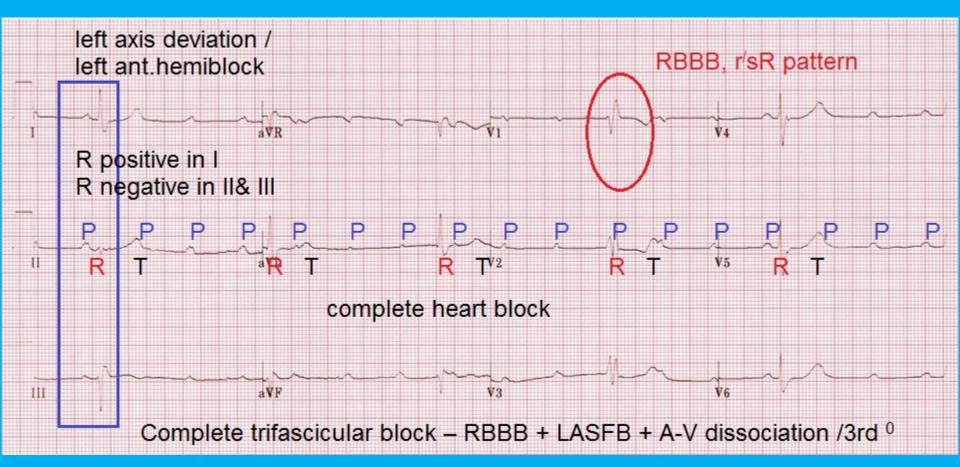


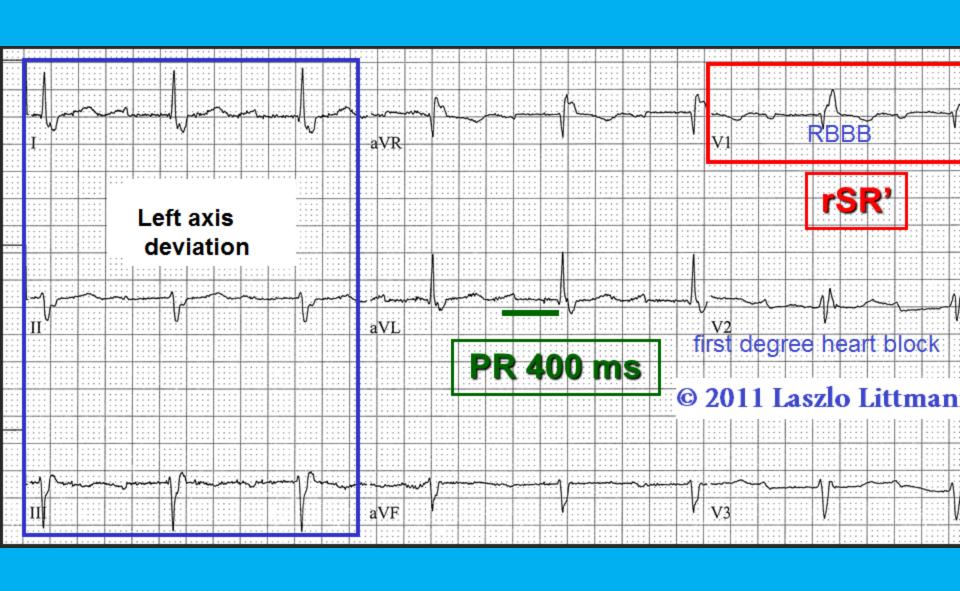














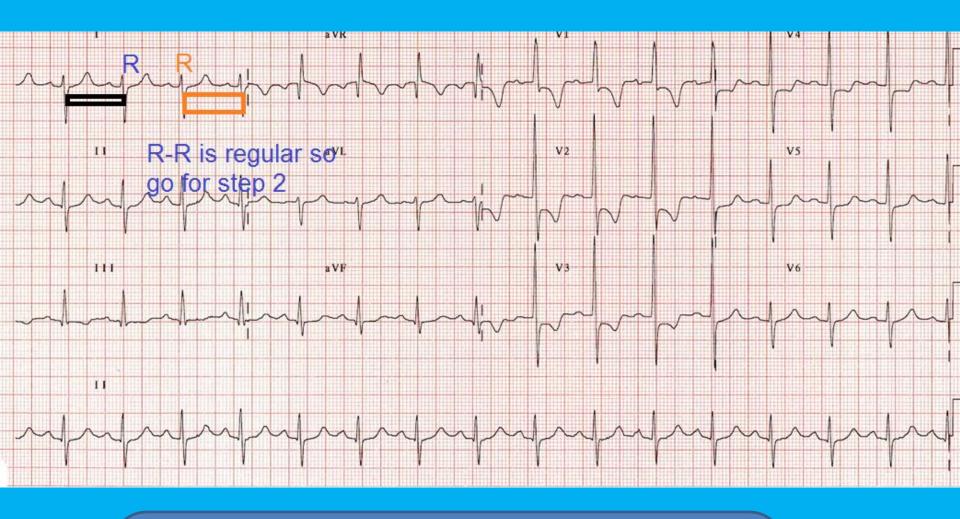
1st seeIn lead IIRhythm

• If Regular --- go next step

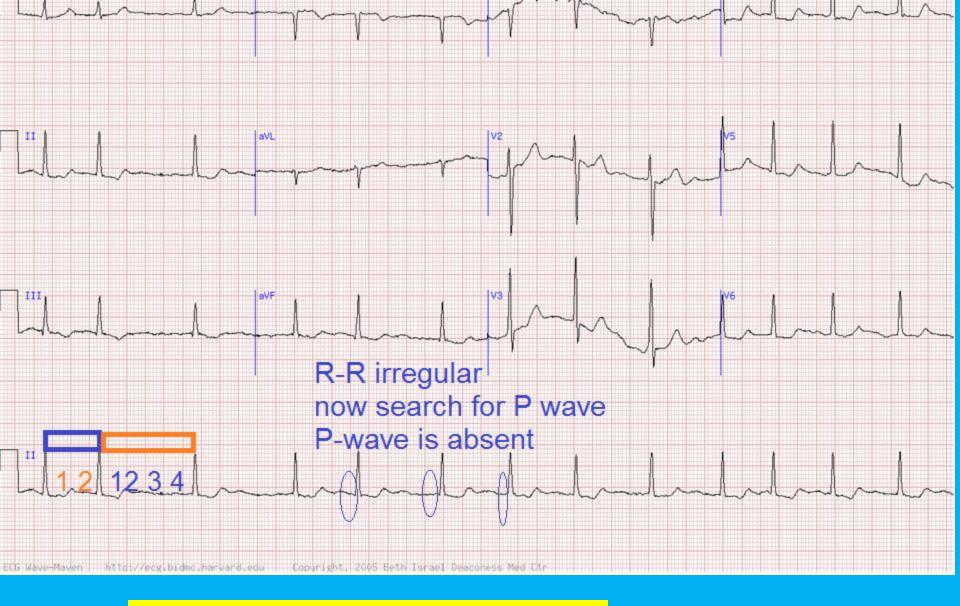
If irregular --it will be AF,

- To confirm look for p wave —if absent —then u r 100%
- if u confused about P wave present or not then look for PR interval,

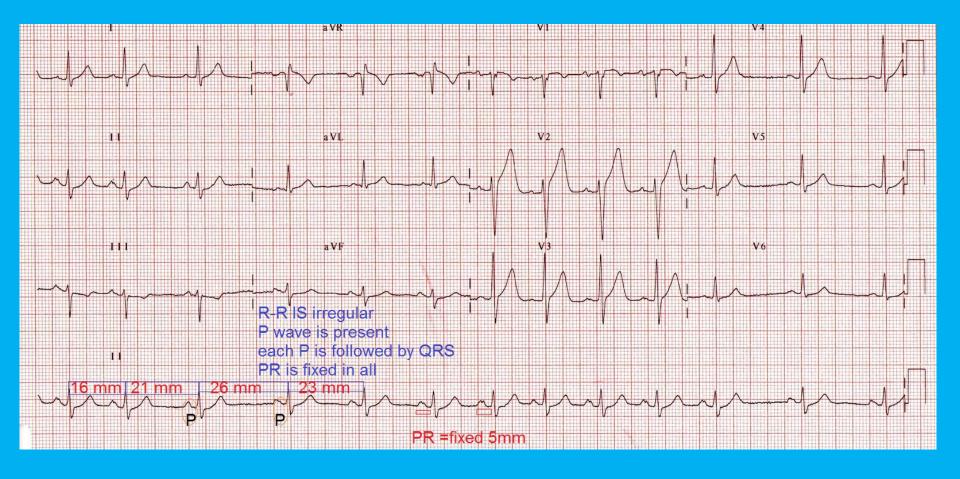
In sinus rhythm P is Isomorphic & PR is fixed before all R in lead II



Here R-R SO go to next step for count heart rate



R-R irregular now search for P wave P-wave is absent --SO it AF



R-R interval is irregular,
PR Interval fixed and P=QRS (every is P
is followed by QRS complex)
So it sinus arhythmia



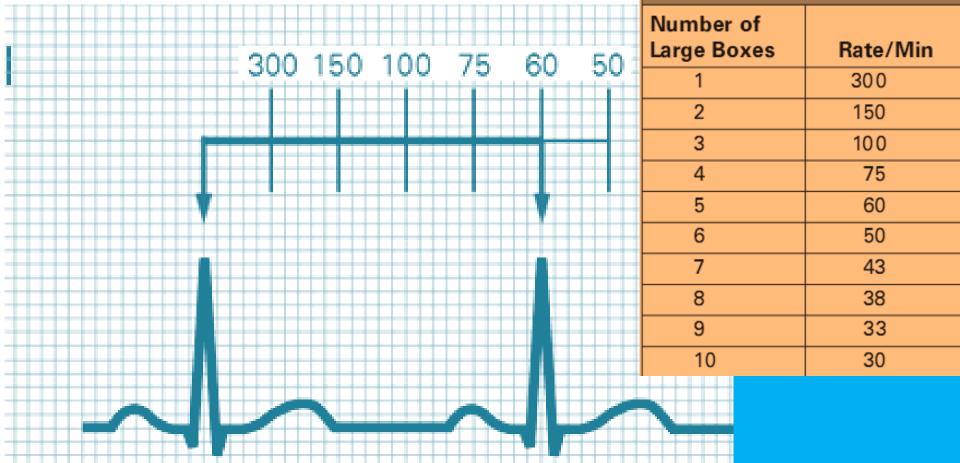
Next see

Heart rate

In lead II

If heart rate is less 40 then

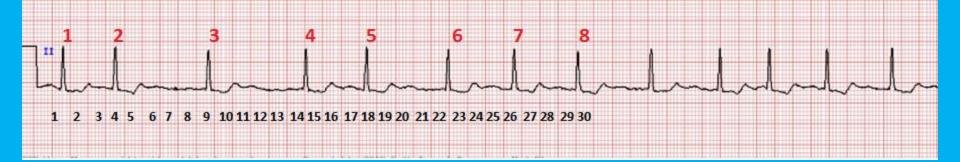
exclude complete heart block
 if your find ECG that
 R-R interval equal and P-P interval equal
 but PR interval is not fixed —then u may
 deal with complete heart block



If rhythm is regular

It calculate the number of R in 6 sec (30 lagre square) and multiply it with 10

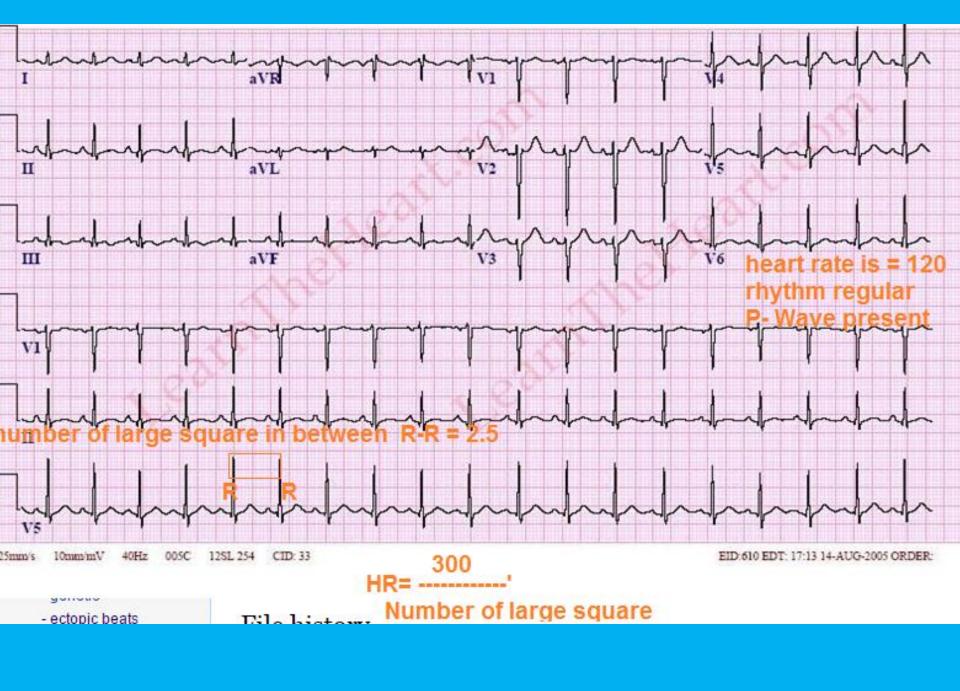
If rhythm is irregular : numbers of R in 30 large square \times 10

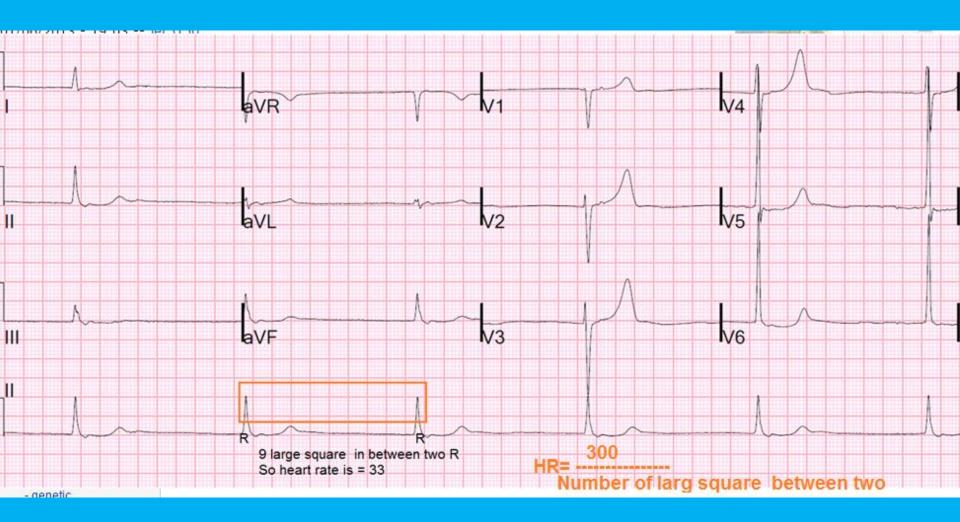


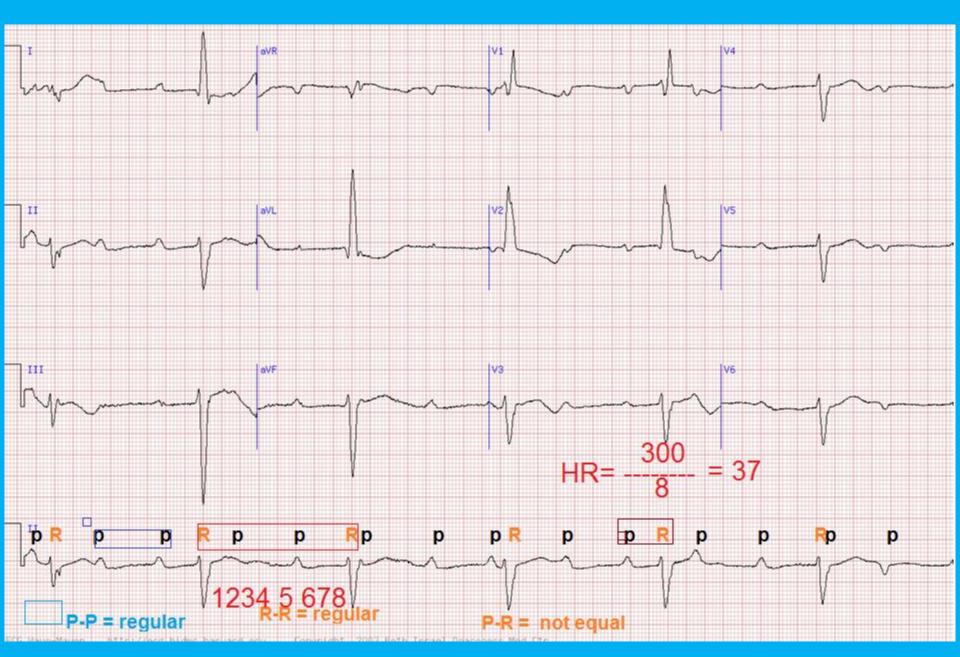
```
there is 8 R in 30 large square
So heart rate is = number of R in 30 large square X 10
= 8 X 10
= 80
```



Fig. 7.5: Irregular R-R interval







Complete heart block

Step =3 MI or ischemia

N	4
	PYI
Τ .	

MI--

Try to find following:

- T inversion
- ST elevation
- Q wave

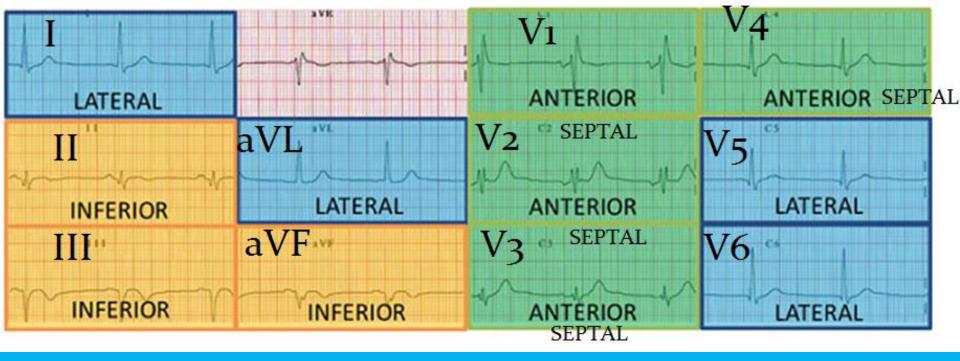
```
1st see -----AVL, I ------
---- lateral MI
```

Then -----II, III, AVF ---- inferior MI

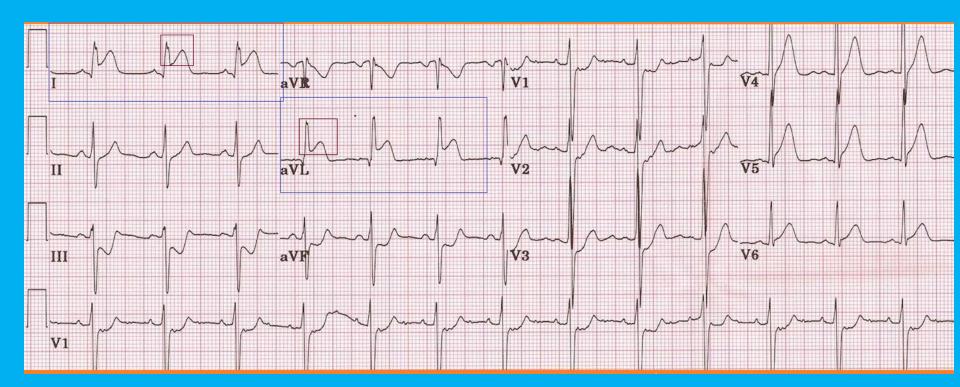
Then----- V_1 --- V_4 ---- antero-septal MI

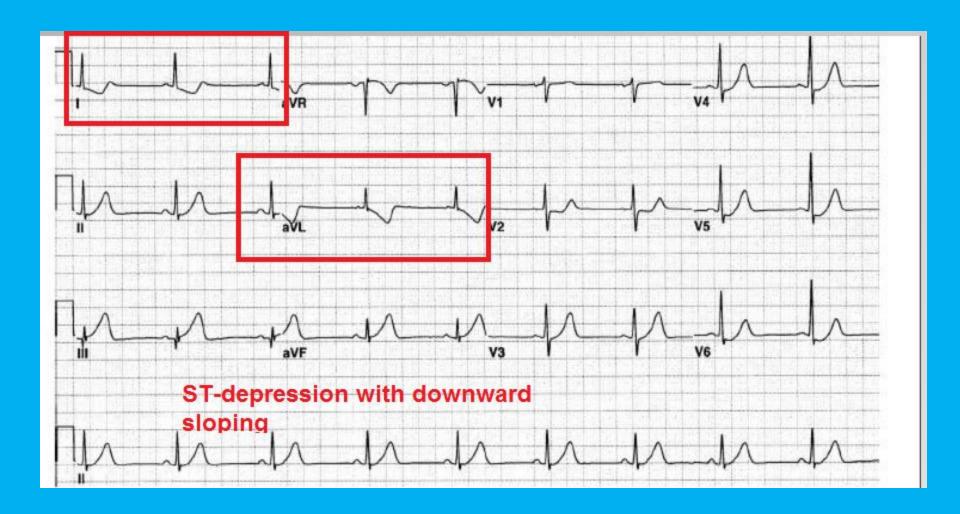
Then ----- V_1 to V_6 -----extensive ant MI

Then -----I, AVL, V_{1} - V_{6}

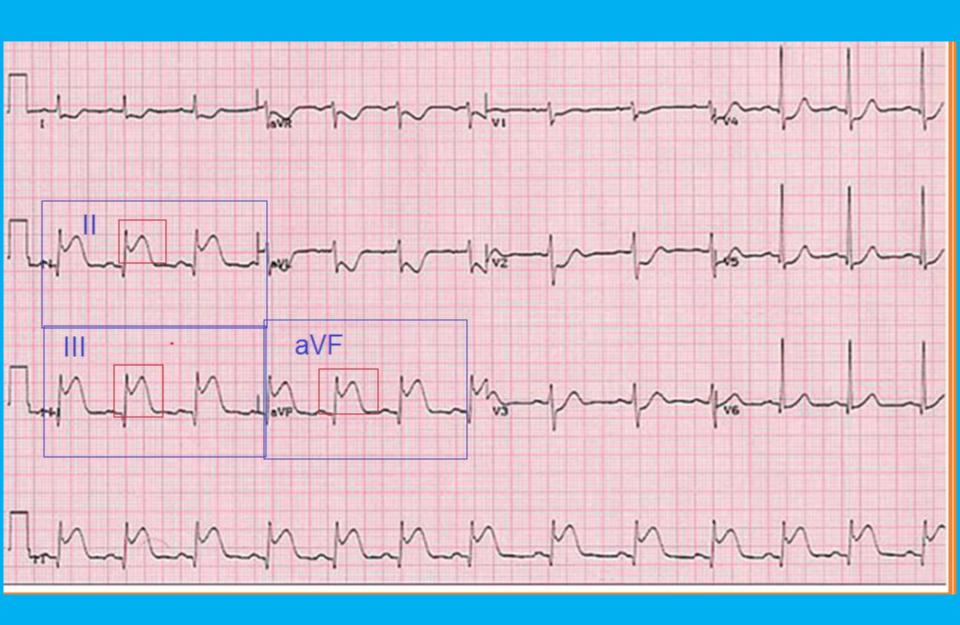


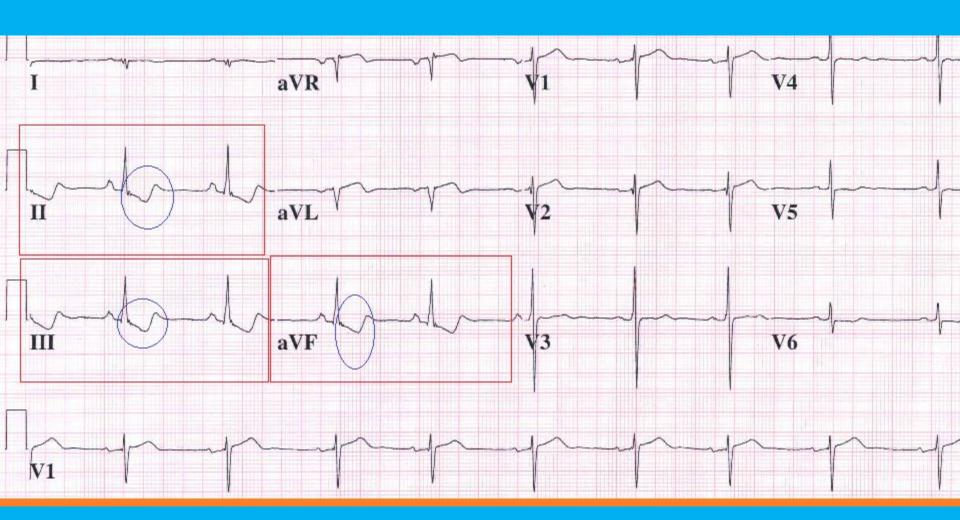
1st see ------ lateral MI & ischaemia



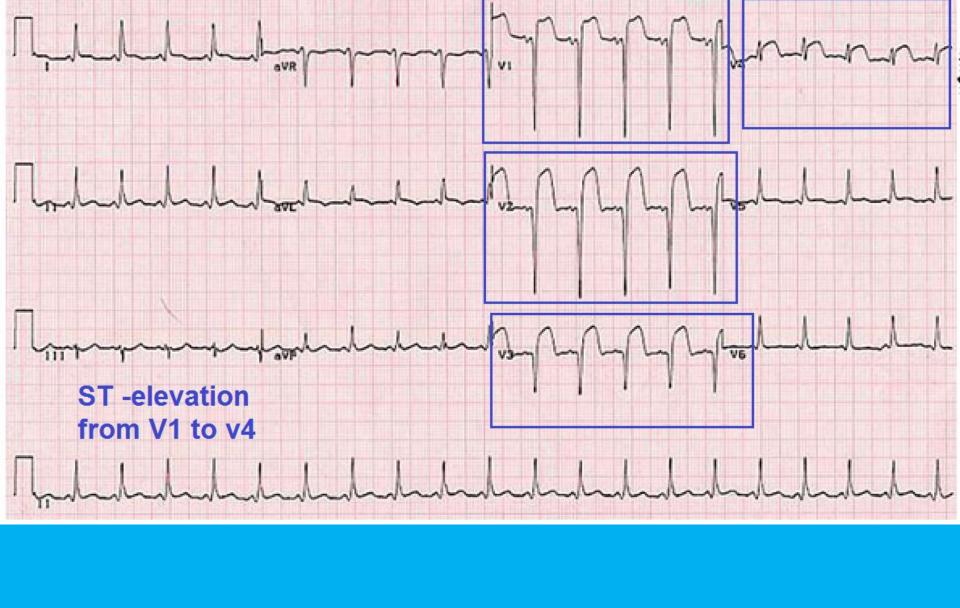


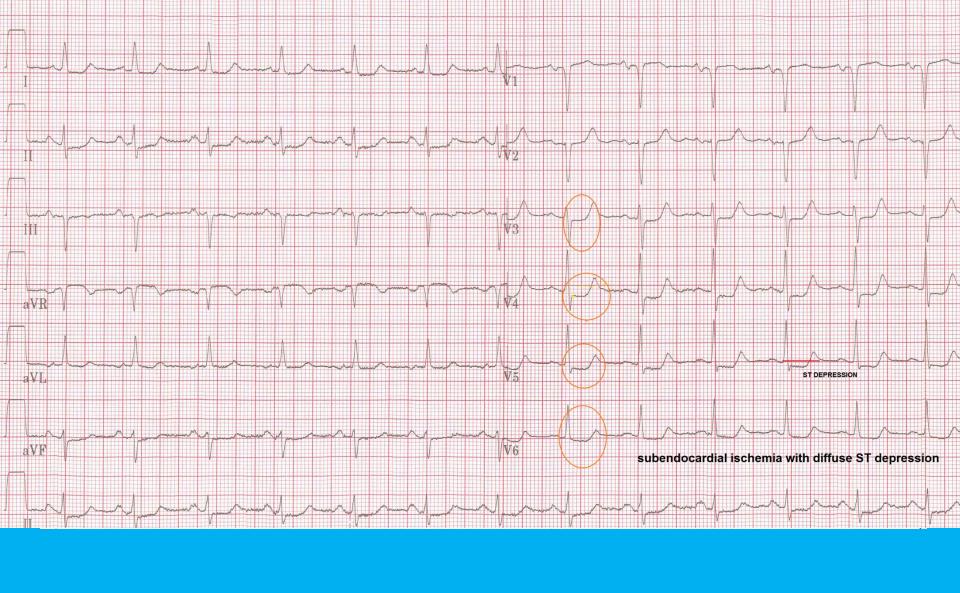
Then -----II , III , AVF ---- inferior MI & inferior ischaemia



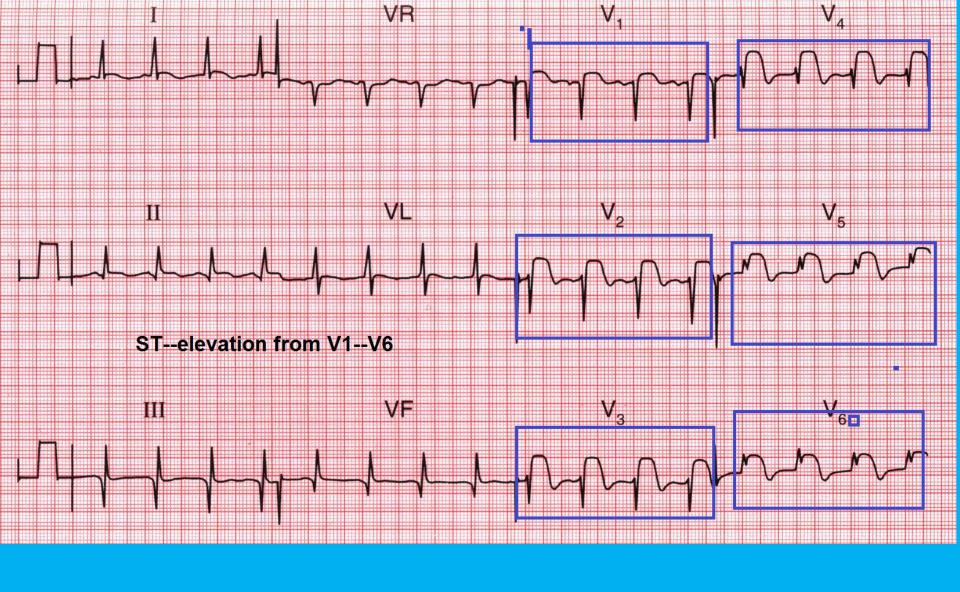


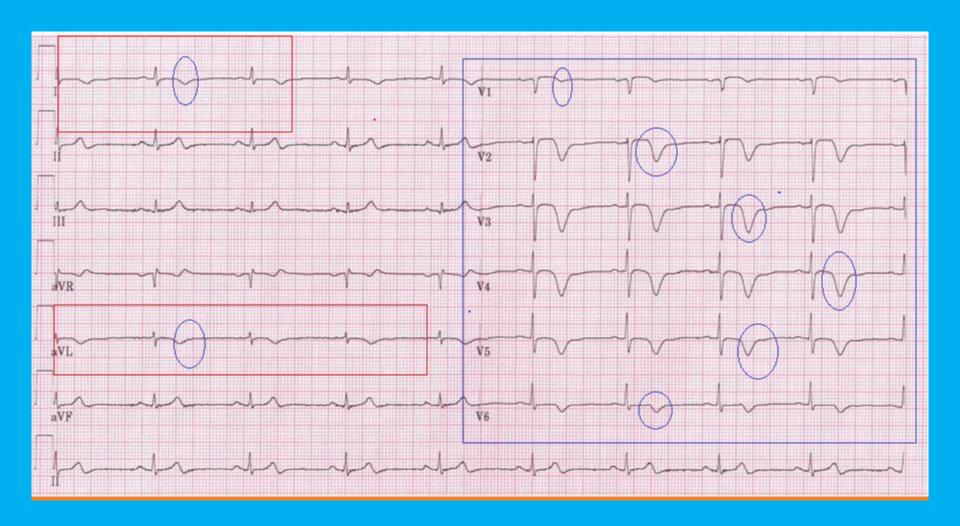
Then-----V₁---V₄---antero-septal MI & ischaemia





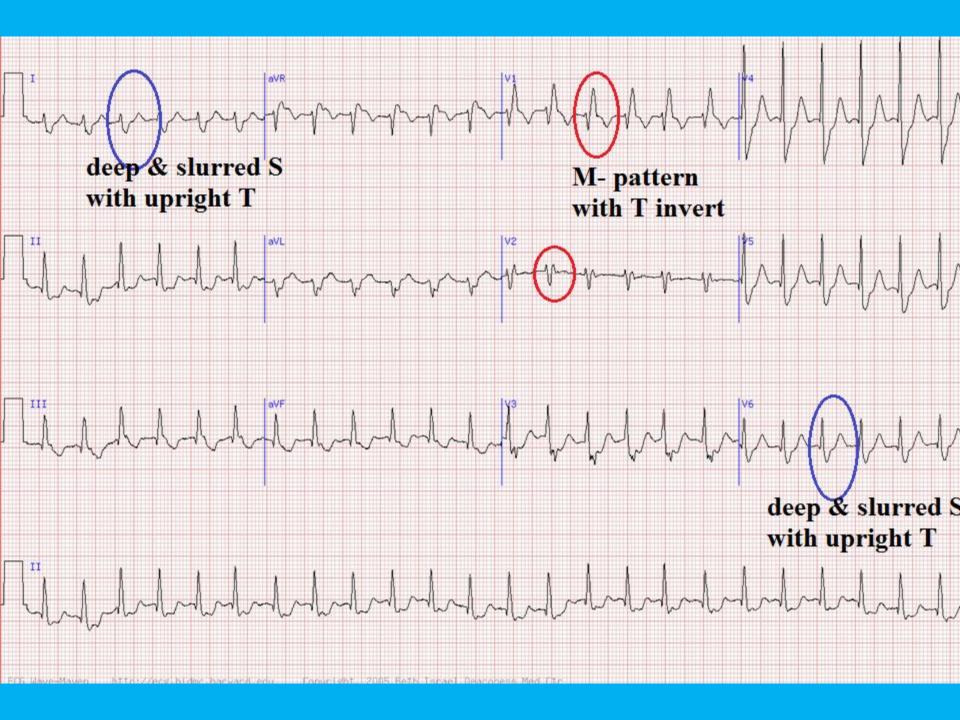
Then ----- V_1 to V_6 extensive ant MI

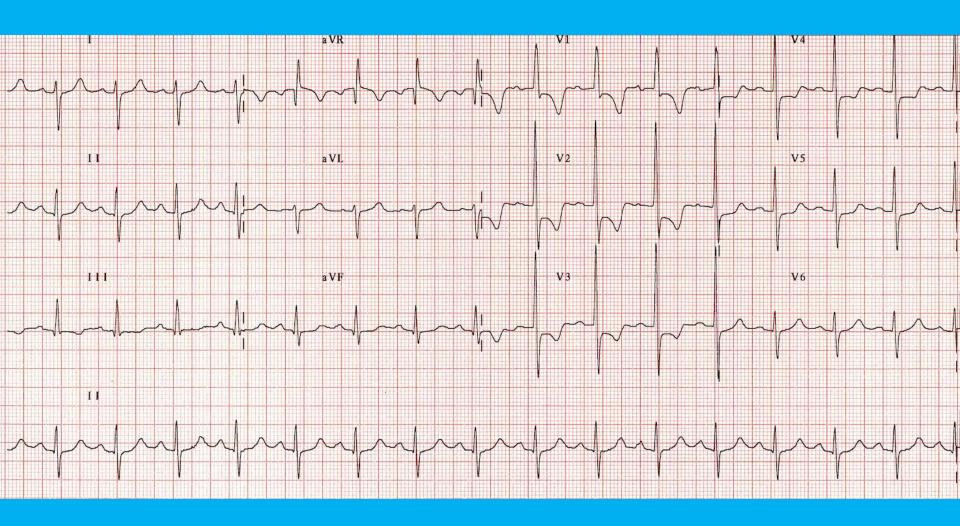






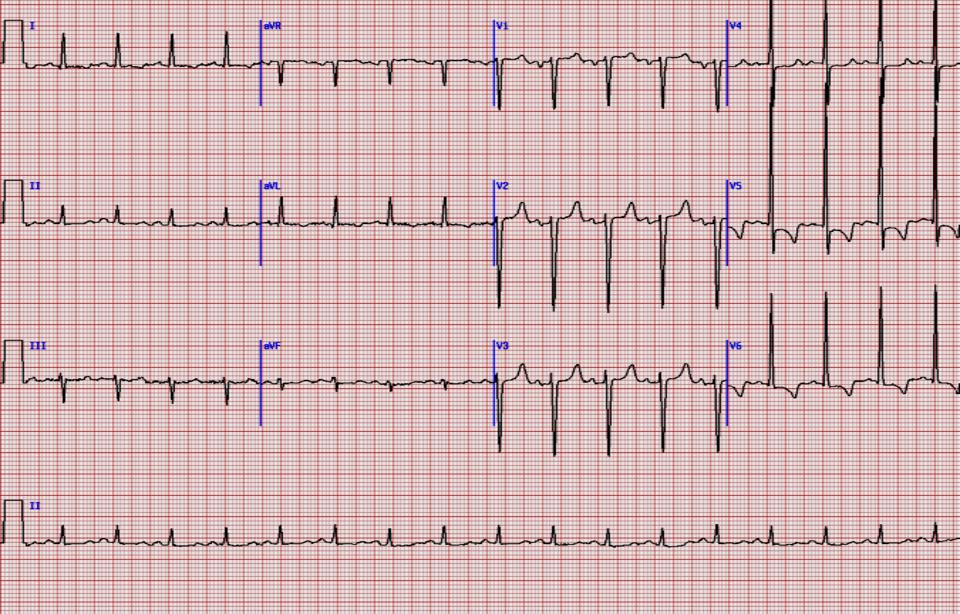
Next see V 1/V 2 (if with T-inversion or ST –depression then **strain pattern**)
Abnormal R or M pattern --- **RBBB**

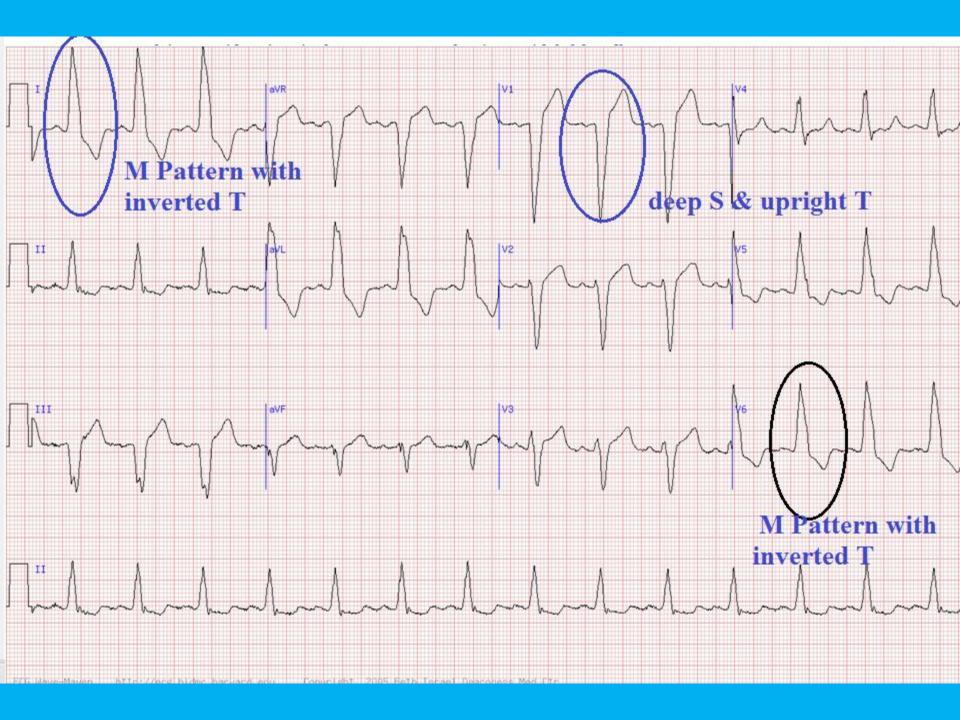






Next see | Tall R--- LVH | (if with T-inversion or ST –depression then strain pattern) | Abnormal R or M pattern --- LBBB







Next see

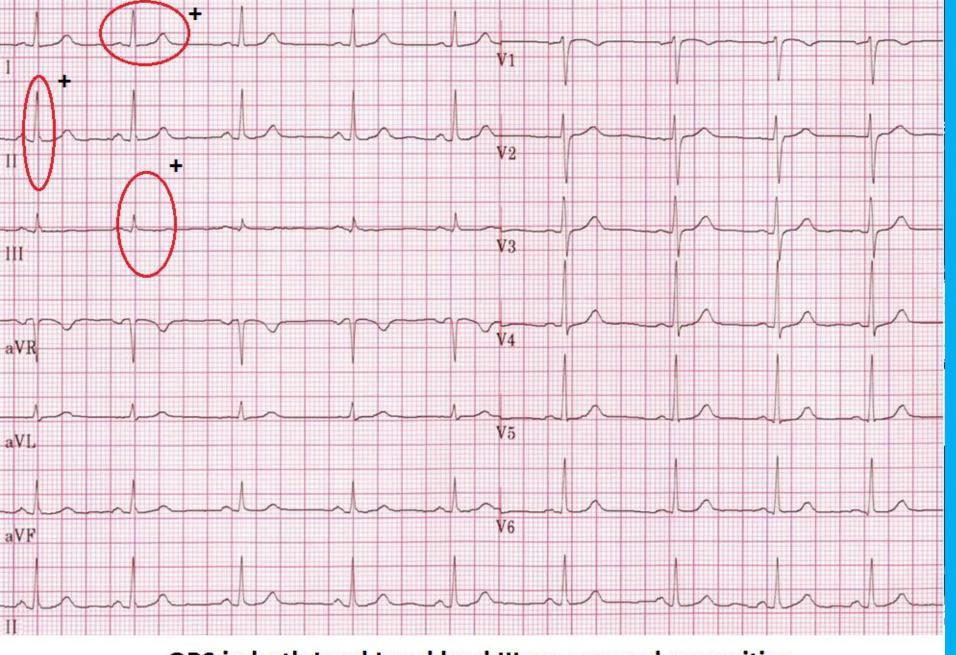
I . II. III

Compare height of R

to see axis deviation

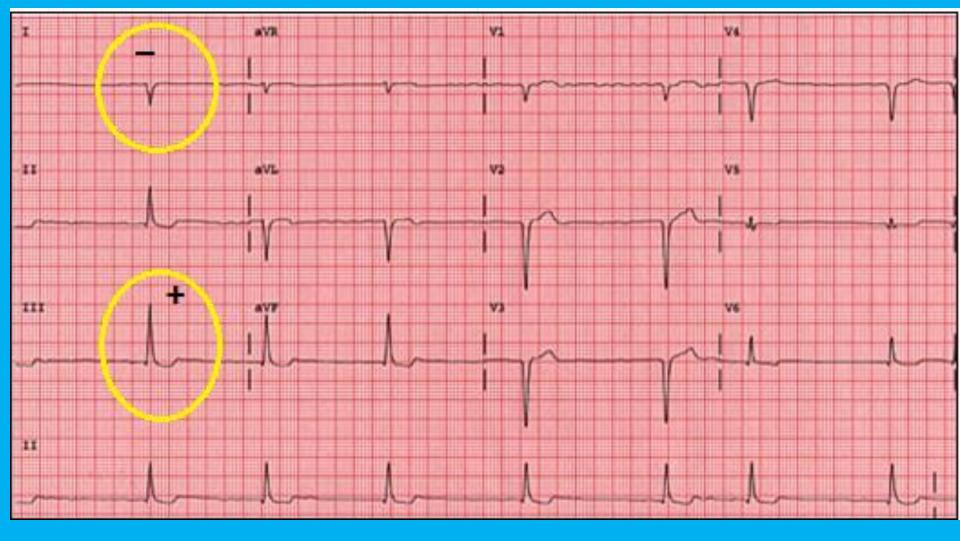
Left axis.... R (+) in I & R (-) II and III

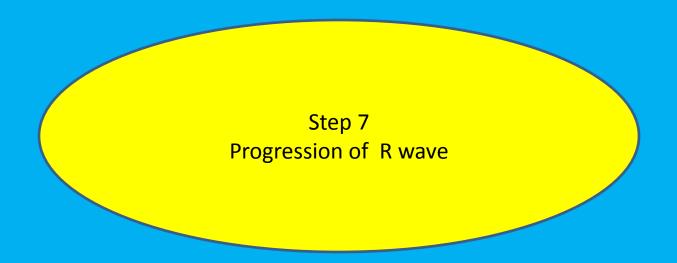
Right axis.... R (-) in I & R (+) II and III



QRS in both Lead I and lead III are upward or positive normal axis





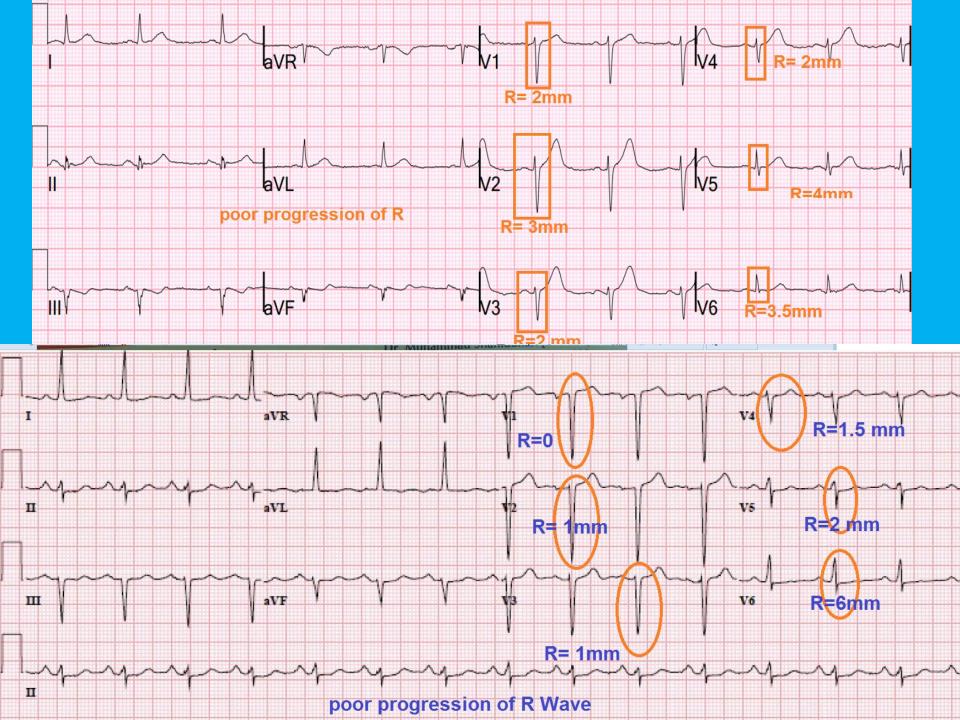


Next see

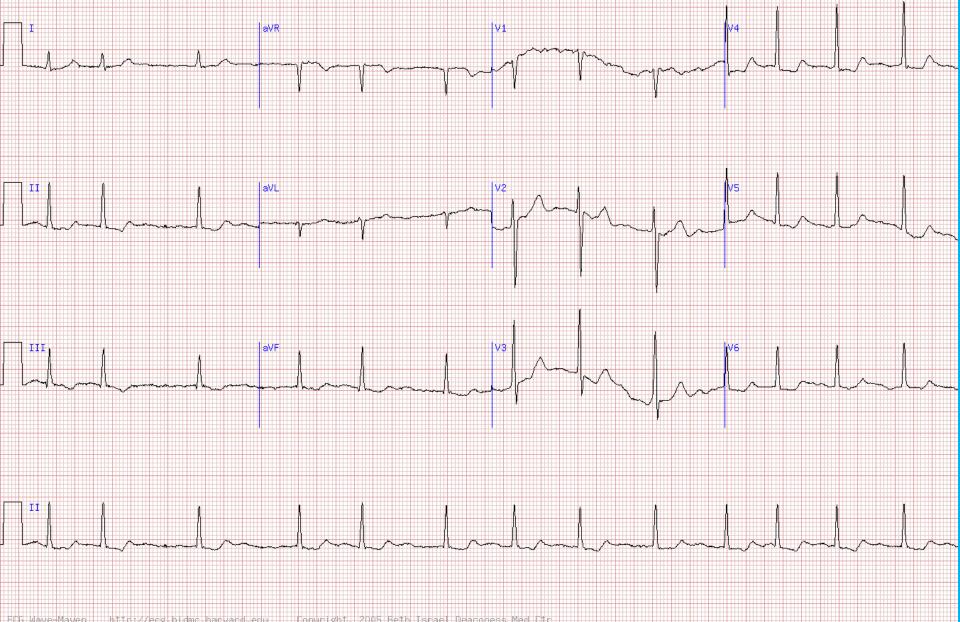
 V_1 to V_6

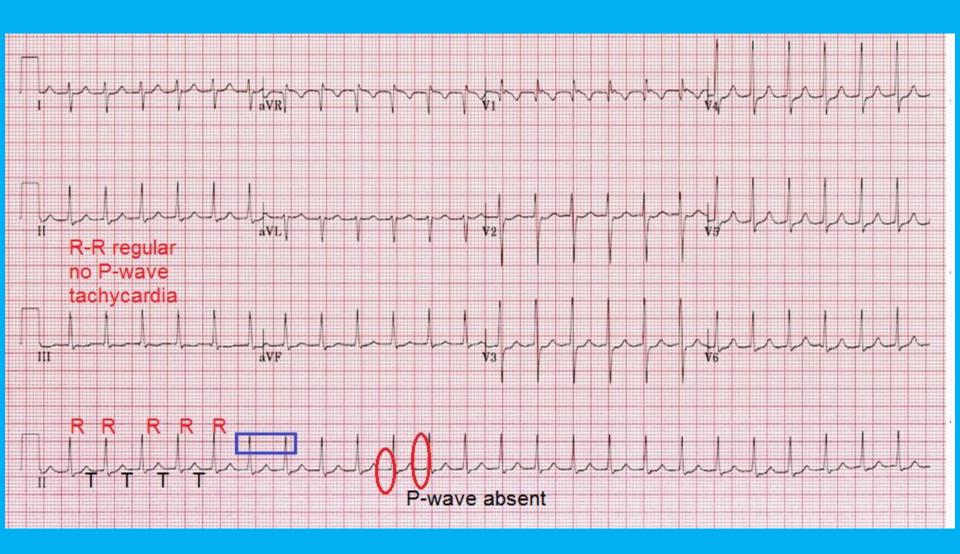
is the height of R is gradually increase from V1 to V6

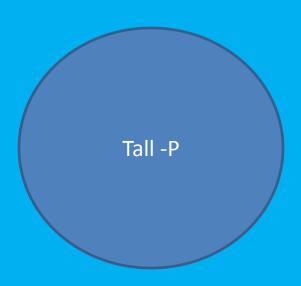
If R in V $_6$ < S in V6 – poor progression of R wave

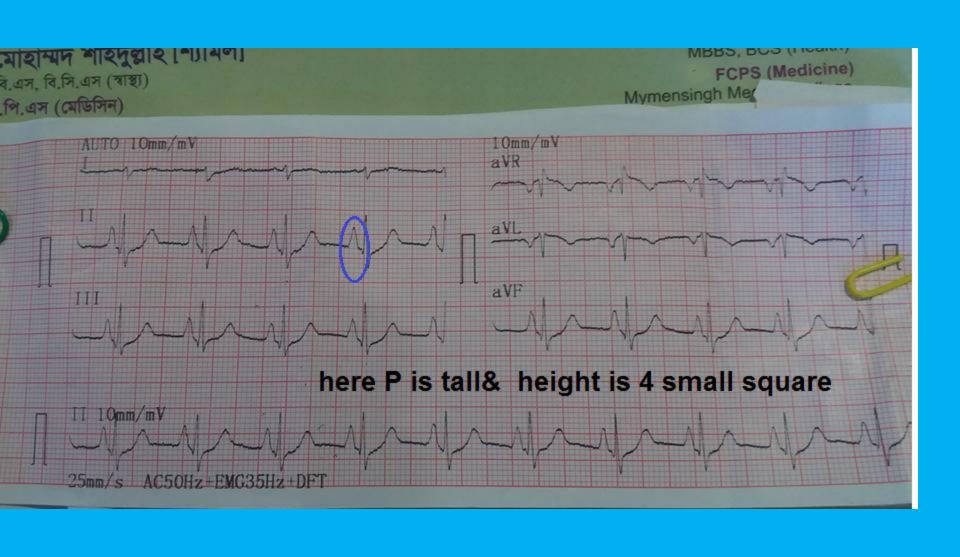


Step 8
Now look for **P** wave
P –present or not
P—morphology tall or wide
PR– interval is fixed or not

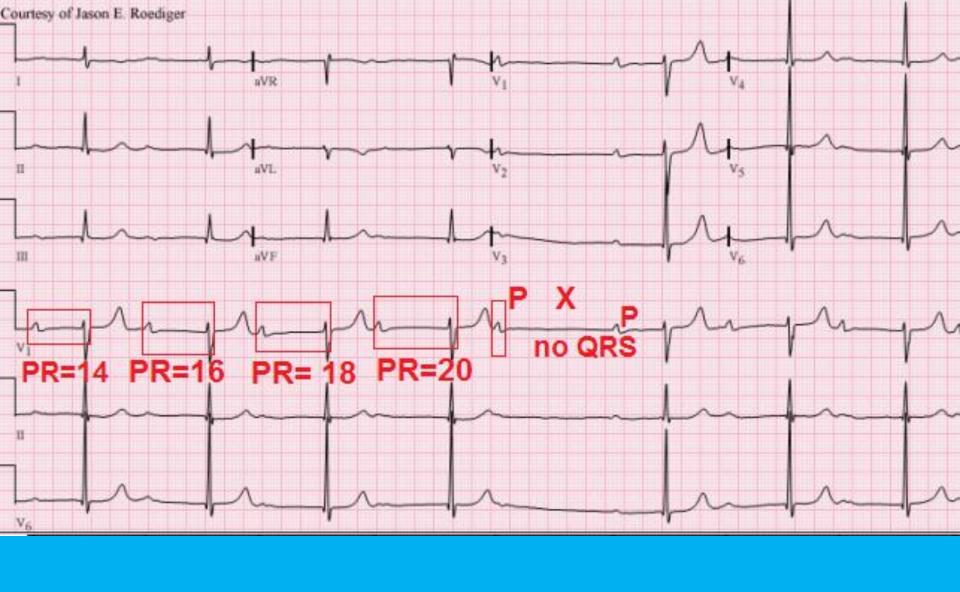


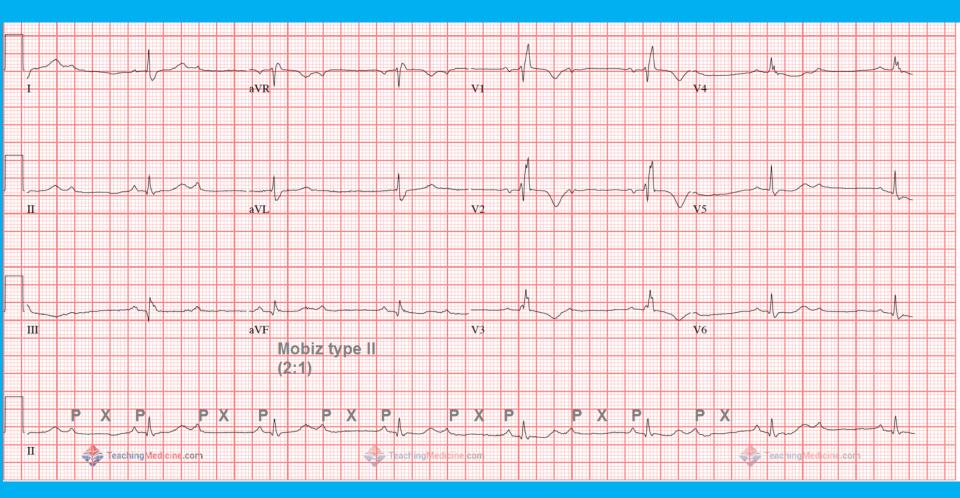


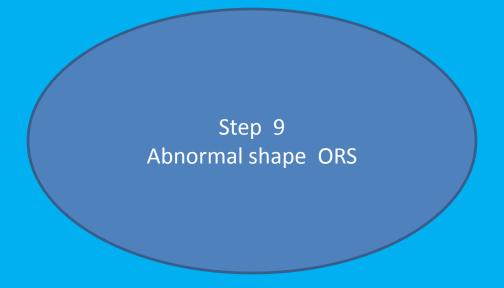


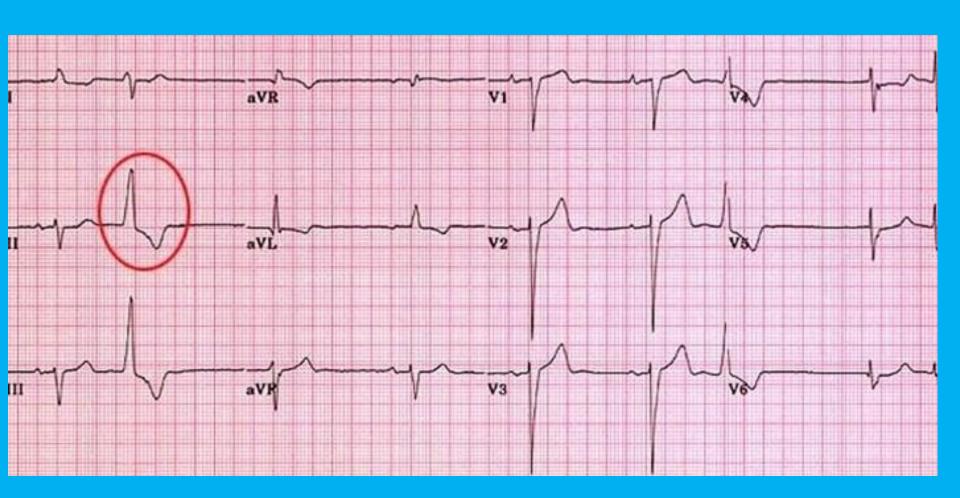


PR- interval is fixed or not Is any P that not followed by QRS complex

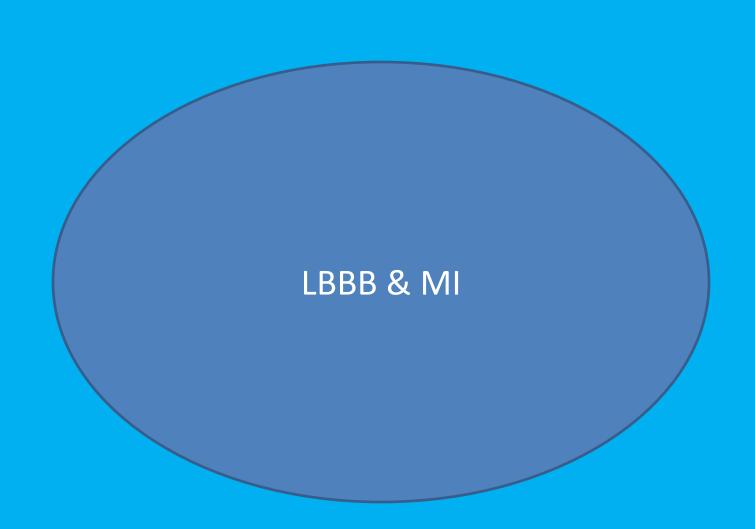






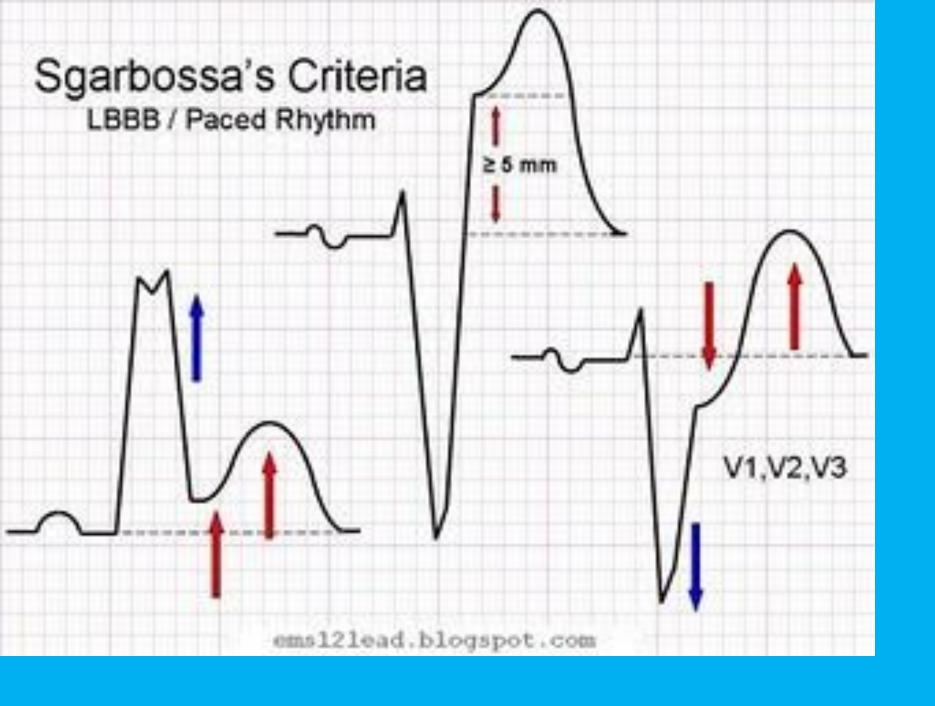


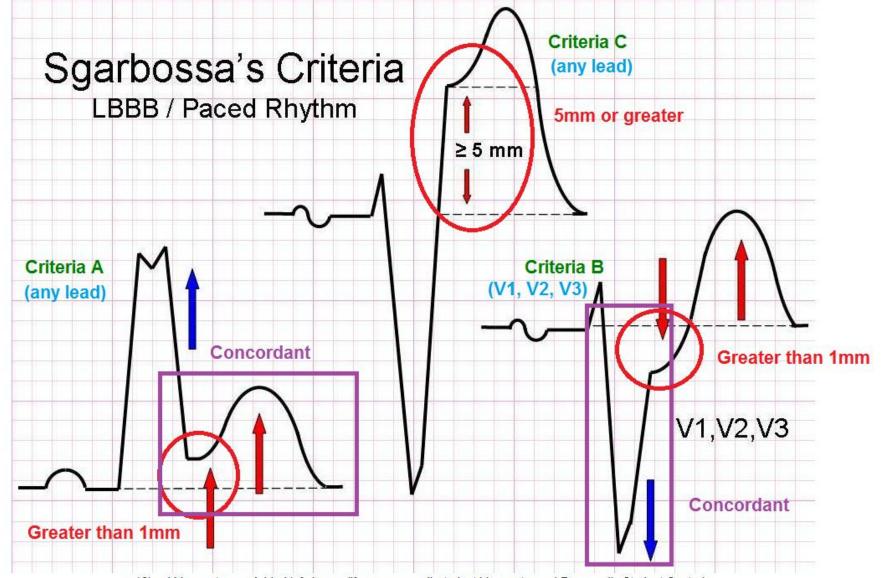




The criteria (Sgarbossa [1]) that can be used in case of a LBBB and suspicion of infarction are:

- ❖ST elevation > 1mm in leads with a positive QRS complex (concordance in ST deviation) (score 5)
- ❖ST depression > 1 mm in V1-V3 (concordance in ST deviation) (score 3)
- ❖ST elevation > 5 mm in leads with a negative QRS complex (inappropriate discordance in ST deviation) (score 2)
- . This criterion is sensitive, but not specific for ischemia in LBBB. It is however associated with a worse prognosis, when present in LBBB during ischemia.[2] At a score-sum of 3, these criteria have a specificity of 90% for detecting a myocardial infarction





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